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APHASIA AND ARTISTIC REALIZATION[1]

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THE subject of this communication lies within the borderland of aphasia; and among the patients suffering from that severe neurological disorder I shall consider only that rare group whose activity before their illness was chiefly devoted to artistic realization. In what measure, and what way, that is altered by aphasia is exactly the question which I shall try to answer, by taking as examples the case-histories of three kinds of artist: writer, musician and painter. Thus my theme is to inquire what happens to productive literary, musical or pictorial activity after the onset of aphasia. Is the deterioration of artistic creation the same in importance in our understanding of the physiopathology of language, of the mental state in aphasia, or even of the psychophysiology of the artist? To that doubtless too ambitious programme, I hope to bring at least some precise material, in some way experimental, drawn from three histories concerning three great artists whom I have been privileged to follow myself over a long time, and whose work before their illness allows a precise comparison between their artistic production before and after their aphasia.

Let us consider first the case of the artist who uses the common tool of language, words and sentences, i.e. the writer. For his artistic creation (prose or poetry) he uses everyone's language. While M. Jourdain, the character of Molière's "Would-be gentleman" was amazed and delighted to know that his language was prose, and that he was thus using prose without being aware of it; conversely the writer makes more or less consciously or cleverly literary works by using everyday language in his own way. The way in which he uses language to dress his thoughts and to express their shades forms the peculiar artistic quality of literary work, and gives it both its individuality and its æsthetic value. That will be destroyed by aphasia.

There is in French literature a sadly famous example of a great poet

[1]Being the Harveian lecture of the Harveian Society, delivered March 17, 1948.

crippled during his literary production by aphasia: Charles Baudelaire, who is perhaps, at least in my opinion, the greatest French poet of the nineteenth century. Suddenly, while his artistic creation was at its height, he suffered from a right-sided hemiplegia with aphasia. We do not know his exact medical history, but we gather from his friends that he was able to say just one word, a curse: "cré nom." He relied upon it to express quite different states of mind. One finds easily there the essential features so well expressed by Hughlings Jackson: the preservation of a "leit-motiv word" (a curse as so frequently is the case), its use with various meanings, sometimes a purposeful one, sometimes an emotional one. But for our purpose one fact is sure: any artistic realization had been since forbidden to Baudelaire, though we do not know whether he ever made any attempt to write with his unaffected hand.

I can afford more precise observations on a contemporary writer, whom I have followed for over ten years, and whose literary position I can only sketch, since he is still alive. When quite young, after he had attained a foremost rank in French contemporary literature by his poems, novels, essays on foreign literature (especially on English literature which he knows perfectly well and which he has interpreted to us in its newest expressions) he was suddenly affected by a right-sided hemiplegia with aphasia. The hemiplegia has remained severe, with a considerable spasticity, interfering with the gait and preventing any movement in the upper limb. Language, at first rather uniformly impaired in its verbal and graphic expressions, comprehension remaining quite good, improved gradually. Today, conversation is possible, but there is a synthetic reformation of the agrammatic type. He went through various phases during the rehabilitation process: at first wide, if not generalized, paraphasia, with a curious recurring utterance—"Bonsoir les choses d'ici bas" —he used to answer in an involuntary and automatic way every time he wanted to say anything. Even the customary "Bonsoir" was replaced by that melancholy expression. Then the "leit-motiv" sentence disappeared during the improvement of his voluntary speech, which was at first poor, slow, difficult and then more definite, despite his painful and at times impatient or disheartened attempts. At the present time, the trouble of synthetic construction remains the major element; the phonetic trouble has almost disappeared and the voluntary evocation of vocabulary is greatly improved. Written language has followed a parallel restoration. It is of course performed with the left hand and there is in it the same agrammatism. On the contrary reading became rapidly possible, although slow. But if he can now, as before, read his favourite books in several languages (and before all English, Italian and Spanish, without any noticeable difference) he can write only sentences where the persistent

trouble of synthetic construction forbids any literary production (at least the one he was used to for he did not employ that conventional agrammatism of some of the young literary schools).

Is the abolition of artistic activity in our patient due to a special deterioration of mental abilities caused by the disease? We have made a special study of that problem many times and we think it is not possible to correlate the inability of this writer to continue his work, with a deficiency of memory, reasoning, judgment or affectivity. His memory is remarkably preserved, of which I can give an example: once, while speaking with him about English contemporary literature, at a time when he was still very aphasic, he had just shown me an autographed book by James Joyce, and he wanted to refer to another author, upon whom he had also written an essay, but whose name he could not find despite his efforts. As I could not make out whom he meant, he stood up and walked uneasily to his library which contained several thousands of English books, and without any hesitation picked out the volume he wanted. It was by Coventry Patmore. His judgment can be evaluated by the way he saw through the press his works which were still unpublished at the time of his aphasia, and more particularly by the following fact: in the course of collecting some essays to be published, one of his friends, who was in charge of the publication, was of opinion that one of the chapters was of a lesser quality. He did not know how to make this understood by the patient without hurting his feelings. He was amazed to see the patient himself, despite his aphasia, realize that inferiority. The pages of the manuscript were turned before him, and at the place of dubious value, his friend asked him whether it should be omitted. As if he had understood, he pointed out the part in question, and said with effort—"not good"—finding at last the way to express his thought. I can add that the æsthetic taste of my patient does not seem to have been impaired by aphasia. Many examples show me that the characteristic trends of his literary personality have not been modified by the illness.

This, then, is an instance of a Broca aphasia in a writer which in spite of its marked improvement, and although it did not noticeably impair the æsthetic sense, judgment and memory, has abolished the possibility of literary artistic realization. The reason lies mainly in that disturbance of literary technique due to disease which converted that delicate artist and subtle grammarian into an agrammatist.

The next case-history concerns a musician. The artist who expresses himself by music uses a language which is not the common language made up of words and sentences. His is the musical language where instead of words the symbols comprise various sounds, united into melodic phrases, which are endlessly varied by the laws of harmony, instrumental

technique and rich orchestral possibilities, just as the word assemblages —sentences of the poetic or prosaic speech—are endlessly diversified by the thoughts of the writer who uses them. To a spontaneous language, which, we are told, can be traced up to Amphion, the sons of man have given by the protean wealth of their sound-combinations unequalled powers of evocation, where each in turn sings joy or pain, pleasure or melancholy. Cleverly elaborated technique allows its extreme differentiation which seems endless. Just as it breaks the poet's calamus, so aphasia ruins Orpheus' lyre.

My second example is that of a famous musician whom I followed for over two years. He belonged to the pleasing class of musicians who have introduced a novel style, an original manner, and have expressed in a particular language a personality which will endure in the history of his art. At the peak of his artistic achievement, rich through an abundant and varied work, already classic, which expresses a delicate climate, Maurice Ravel is struck down by an aphasia. His aphasia is more complex than the writer's: it is a Wernicke aphasia of moderate intensity, without any trace of paralysis, without hemianopia, but with an ideomotor apractic component. The cause, though indefinite, belongs to the group of cerebral atrophies, there being a bilateral ventricular enlargement; but it is quite different from Pick's disease. Oral and written language are diffusely impaired, but moderately so, without any noticeable intellectual weakening. Memory, judgment, affectivity, æsthetic taste do not show any impairment to repeated tests. Understanding of language remains much better than oral or written abilities. Writing, especially, is very faulty, mainly due to apraxia. Musical language is still more impaired, but not in a uniform manner. There is chiefly a quite remarkable discrepancy between a loss of musical expression (written or instrumental), and musical thinking, which is comparatively well preserved. With the help of two musicians, a favourite pupil of the master and a neurologist with great musical ability we could study as precisely as possible musical tune recognition, note recognition (musical dictation), note reading and solfeggio piano playing, and dictated musical writing (copied or spontaneous). I apologize for giving such an analysis, but it seems to me essential in respect of the value of such a case-history.

Recognition of tunes played before our musican is generally good and prompt. He recognizes immediately most of the works he knew, and anyway he recognizes perfectly his own works. That recognition is not a vague one, for he is able to evaluate exactly rhythm and style as shown by the following facts. He immediately notices the lightest mistake in the playing; several parts of the "Tombeau de Couperin" were first correctly played, and then with minor errors (either as to notes or rhythm).

He immediately protested and demanded a perfect accuracy. When playing the beginning of "La pavane de ma Mère l'Oye" which contains two exactly similar bars, one was omitted. The patient immediately stopped the pianist. He succeeded in explaining, in his halting speech, that the first bar was to link with the preceding part. The same is true for rhythm: if played too fast, he protests and has the music played again with its exact rhythm. Another remark: during these studies on musical interference of aphasia, my piano—because of the dampness of the winter—had become somewhat out of tune. The patient noticed it and demonstrated the dissonance by playing two notes one octave apart, thus showing again the preservation of sound recognition and valuation.

On the contrary analytic recognition of notes, and musical dictation, are quite faulty, or seemingly so, since he could name only some notes hesitatingly and with difficulty. His numerous mistakes are due, very likely, to aphasia itself, and to the difficulty of finding the name of a note, a trouble exactly similar to name designation for common objects. The fact that reproduction of notes played on the piano, without giving their name, is quite good, seems to confirm this opinion.

Note reading is extremely difficult. From time to time a note is read exactly. Most often reading is impossible. The same is true for solfeggio. The trouble of name-finding may partly explain the failure. But there is something more, since piano playing is almost impossible after reading. A component due to apraxia supervenes therein. Anyway a quite definite discrepancy is noted between deciphering musical signs, and their visual recognition. If an analytic deciphering is almost impossible, on the contrary the patient is able to recognize at first glance whatever piece he has to find, and that without any error.

Piano playing is very difficult, since in addition to difficulty in reading, our aphasic patient has to search for the location of notes on the keyboard. He sometimes misplaces notes without being aware of it. For instance he plays the *mi-mi* instead of the *do-do* arpeggio, and plays it again and again, until his fingers are placed on the proper keys. He plays scales quite well, both major and minor ones. Diesis and flats are well marked. There is just a praxic difficulty. He can play with only one hand (the right one) the beginning of "Ma Mère l'Oye." With both hands, he cannot decipher. He needs many exercises to play in that way. In spite of numerous exercises during a whole week he cannot succeed in playing the beginning of the "Pavan," even with separate hands. On the contrary he has a greater performance ability when he plays by heart pieces of his own composition. He suddenly gives a right idea of the beginning of "Le Tombeau de Couperin" which is, however, too difficult to finish. Seven or eight bars are played almost perfectly, and he plays

them, transposing to the lower tierce, without any error. When attempting an unknown piece he finds a much greater difficulty: he cannot play more than two or three notes of a piece by Scarlatti, which he did not previously know.

Musical writing is very difficult, although better preserved than plain writing. He writes dictated notes slowly and with numerous errors, but copying is almost impossible and requires from the patient enormous effort. On the contrary, writing by heart a portion of his "Entretiens de la belle et la bête," though difficult and slow, is better performed than the other tests. Notes are better and more quickly placed, and he seems mainly disturbed by writing apraxia. Singing by heart is correctly performed for some of his works, but only if the first note or notes are given. He says that tunes come back quite easily, and that he can hear them singing "in his head." Musical thinking seems comparatively better preserved than musical language itself.

Though all artistic realization is forbidden to our musician, he can still listen to music, attend a concert, and express criticism on it or describe the musical pleasure he felt. His artistic sensibility does not seem to be in the least altered, nor his judgment, as his admiration for the romantic composer Weber shows, which he told me several times. He can also judge contemporary musical works.

Thus, in our musician, because of aphasia, and as already mentioned, because of a simultaneous apraxia, musical reading, piano playing, use of musical signs are much more impaired than expression and recognition of musical themes. Severe disturbance of realization, and difficulty of expressing a relatively preserved musical thinking, while affectivity and æsthetic sensibility are almost intact, are the main features of our composer's case-history. They explain why his work has been completely arrested by his cerebral affection.

And now here is the painter. The artist here does not use for his creation oral or written language. From his visual perceptions, and according to the way his hand can transcribe them, through drawing and colours, a particular æsthetic pleasure will emerge through the magic of a figurated art. From sensory images taken out of nature, and from the observation of the marvellous playing of light upon forms, a personal vision is suggested, in which are brought to life the expressions of a varied poetry, from the moving grace of a face, to familiar or grand life, from the charming melancholy of the luminous haze on a landscape to the merry glow of a bunch of flowers. What will aphasia do against the realization of that living and coloured architecture? If it destroyed literary language in the writer, if it stopped sound expression in the musician, it leaves untouched plastic or figurated realization.

The painter who represents my third example is one of the most representative artists of the French contemporary school, because of the originality of matter, technical qualities of realization, and intense individualism of each of his works. He expressed the poetry of the Normandy coast, the glowing beauty of flowers or marine life, and the richness of feminine flesh. Poet and symphonist as much as a painter, he creates a climate where personal sounds vibrate, and gives to plastic art numerous resoundings, at first of a different nature: one often feels the bitter and sensual poetry of Baudelaire, and at times also the delicious polyphonic music of Debussy, mixing by a kind of sorcery their different languages to form his coloured magic. His pictorial art is far from being only a purely sensorial expression; it contains numerous intellectual and affective components, which, however, are untouched by aphasia.

When he was 52, he was suddenly deprived of language after two short and transient aphasic spells. His aphasia is of the Wernicke type, without any phonetic trouble, without any right-sided hemiplegia, but with a slight hemianopic defect. Spoken language is much impaired, paraphasia is striking, voluntary vocabulary finding much disturbed, but understanding remains quite good because mainly of an intuitive grasp of the general meaning of conversation. Written language is impaired in a parallel way for reading as well as for writing. Writing is slow but has undergone no deformation. Intellectual impairment is not to be found. Although oral or written conversation became very difficult especially when concerned with the æsthetic problems in which he used to be interested one can easily realize that memory judgment and taste are not at all impaired. But his always sharp affectivity is still sharper: he is irritable, sad, and disquieted by his illness, and he increases the isolation to which he was prone. His artistic activity remains undisturbed, even at a time when his aphasia had not yet diminished very much. He stopped drawing only for a short time when an apraxia—fortunately transient—supervened.

Artistic realization in our painter since his aphasia remains as perfect as before. According to connoisseurs, he has perhaps gained a more intense and acute expression. If one tries to analyse his production before and after his aphasia, one cannot find since his language deterioration any mistake in form, expression or colour interpretation. There are no changes in the interpretation of sensorial data, no technical failure; neither is there any disturbance of thought. I am of the opinion that he lays emphasis on thematic characteristics with a poetical strength, in a completely unaltered manner, and that since his illness he has even accentuated the intensity and the sharpness of his artistic realization. Moreover, his activity and production have not slowed down, and he

seems to work with the same speed. Perhaps, because of new affective conditions, his works are less regular. His preferred themes are the same without any duplication of the past ones. His inventive ability, his spontaneity seem untouched. Perhaps he is more prone, however, to use a previous model, or a discarded sketch. Anyway, his individuality has not been altered at all, as far as artistic realization is concerned, and every critic can discern in all of his works the hand of the artist he used to know.

It seems that our painter's artistic production goes on as if nothing had happened, and that in him the aphasic and the artist live together on two distinct planes. As he says himself when congratulated upon his artistic activity: "There are in me two men, the one who paints, who is normal while he is painting, and the other one who is lost in the mist, who does not stick to life. . . . I am saying very poorly what I mean. . . . There are inside me the one who grasps reality, life; there is the other one who is lost as regards abstract thinking. When I am painting I am outside my own life; my way of seeing things is even sharper than before; I find everything again; I am a whole man. Even my right hand that seems strange to me, I do not notice when I am painting. These are two men, the one who is grasped by reality to paint, the other one, the fool, who cannot manage words any more." Is it possible to describe better than our painter, with his faulty speech, the discrepancy he felt within himself between his speech abilities and expression through his representative art?

This last example shows—in contrast to what happens to the writer and the composer when deprived by aphasia of their artistic creative power—that a perfect preservation of pictorial artistic realization can occur despite a severe and persistent aphasia.

These are the facts I wanted first to analyse in some detail, to give all the data about the problems they suggest. We cannot think of drawing from these three histories all the conclusions they permit, so inexhaustible is psychological investigation in aphasia, furthermore, our goal is quite different.

It remains that three great artists are incapacitated by aphasia, as far as verbal and graphic expression is concerned. But concerning artistic realization they behave quite differently. Artistic production, completely preserved in the painter, is abolished in the writer and in the composer. These facts seem so definite that they do not need a long discussion, which could be endless, since neither Ruskin in England, nor Taine in France, have exhausted æsthetic problems. More modestly and more practically we simply want to stress what can be learned from these three cases as to the nature of intellectual impairment in aphasia, and its consequences in artistic realization, depending on the different nature and means of expression of the three arts.

Let us first remark, to avoid criticism about methodology in seeking to compare facts of too different a nature, that we are perfectly aware of the differences between the aphasias of our various artists. But has man ever seen two exactly identical aphasias? That does not depend only on the differences between the lesions (we do not consider anatomical or topographical conditions since our analysis is purely a functional one). We do not misjudge the various influences of somatic lesions on physiological and psychological processes which lie beneath language, but we believe that everyone's language is the expression of a complex whole, that goes beyond language itself, and expresses a totality and an individuality. Aphasia more or less respects that personality, as far as it remains unaltered by psychic troubles. These individual differences will help to differentiate each aphasic patient's language, even if gravely altered. Anyway the aphasia is different in each of our three cases. The writer has a Broca aphasia, the musician and the painter have a Wernicke's aphasia, complicated by a permanent apraxia in the composer and a transient one in the painter. But we know that if the phonetic troubles which characterize Broca's aphasia have a definite physiopathological and topographical value, as far as neurological interpretation is concerned, it is not enough to differentiate the aphasia with which it is associated. Language troubles are not of a different nature because a motor trouble which has to do with the organs of speech supervenes in these cases. That allows a comparison with the cases of the musician and of the painter whose language was impaired in a similar way, but without any phonetic syndrome. More important at first sight is the fact that our composer's aphasia was accompanied by apraxia, the rôle of which we have stressed in some of his troubles with musicial expression, especially instrumental execution. On the contrary apraxia has nothing to do with his other defects in musical language. In reading, and in musical dictation, apraxia does not interfere, and cannot be blamed for the defect in artistic realization. Our musician might have remained a great composer, even if unable to play or to conduct his works. Is it not a similar example on a purely sensorial plane to the one given by Beethoven, deaf, going on with his work, without being able to hear it, or by Milton, finishing his admirable poetical work which he could not read?

The main fact is that our three aphasic artists have in common a language trouble that spares their personality, especially its æsthetic components that does not alter at all their sensibility, their memory, their judgment, or their taste, and where means of expression are much more altered than understanding. On the contrary they differ in that the writer and the composer are, consequently, annihilated as to their artistic production, while the painter is not affected. Although the same sensi-

bility and æsthetic taste remain, although he is able to read his favoured poet, even in a foreign language, our writer is no longer able to describe orally or by writing his emotion or his own artistic ideal. Although he possesses a relatively intact power of musical thinking, with excellent rhythm and recognition of style, and an unaltered æsthetic sensibility, our composer canot give us any longer the expression (written or melodic) of his delightful musical language. As for our painter, although he explains only with imperfect and vague words the aims of his research for colour values and the significance of his plastic figuration, he continues as well as before giving us speaking pictures as beautiful as ever.

This is the main fact that comes out of the comparison of our three artists. If, as Jackson said, one does not try to define whether intelligence is altered in aphasic patients, but only what form of intelligence is modified, one can say, doubtlessly, that the work of art, or at least its realization—that essentially intellectual manifestation—is not identically altered in aphasic artists. Since their similar language disturbance does not show any difference that may explain the fact, it is in the different nature of their art, and in the means they use to express themselves, that the reason of it is to be looked for. Being especially disabled in their expressive abilities by aphasia, as we have seen, our writer and composer are not able to express themselves by literature or music, for their technique is based on language and we have no reason to wonder at their failure in artistic realization. But since the painter does not suffer from his language deterioration, we have to consider the insignificant part played by language in plastic realization.

So we come to investigate technical means of the arts we consider in their relation with language. Art may be described as the expression of an individuality by way of appropriate technique. We have seen the almost complete preservation of each artist's individuality. Let us consider the differences between their technique.

Literary art is entirely dependent upon language in its outward expression. Nobody can deny it. Highest thinking, deepest sentiments are just immaterial facts of consciousness, if they are not clad in the flesh of language. When considering an aphasic patient's behaviour, while he uses his emotional or volitional speech, one cannot wonder at the aphasic writer meeting a considerable amount of difficulty in literary expression. Imprecision of aphasic speech, difficult adaptation of thinking to verbal or graphic expression, failure or slowing of realization are among the most important causes of failure. Difficulty in reaching abstraction for a writer who would not stick to material expression of a schematic tale is another main stumbling-block. Furthermore, if there is a synthectic disturbance, i.e. agrammatism, one may realize the presence of an invincible difficulty.

because of the chief importance of grammatical construction and style in literary realization. The aphasic patient, with the involuntary use of ready-made sentences unadapted to what he wants to express, shows in some way a negative picture of literary art. One can say that his thinking itself is not noticeably impaired; realization of linguistic form, on the contrary, will remain quite unadapted. The aphasic patient, as Hughlings Jackson said, "is lame in thinking," one could add "he is lame in literary writing."

For the composer, who too has to master a language to express himself, the same is true. Musical language is made up of signs, where sounds represent a mere symbolism, but where the modes of musical phrases, with their intentional descriptive or affective meaning, represent a superior symbolism. Melodic forms are cleverly placed according to more or less conventional, arbitrary or individual patterns, in which may be recognized, superimposed on his thinking, the genius or technical ability of the author. Furthermore graphic transcription of a musical work needs a scriptorial expression similar to literary writing. That shows how many factors, of various levels, make artistic realization, as expressed in a musical work, and how such technique will be altered by aphasia as easily as the technique of literary expressions since we deal here also with an artistic expression through a language. That explains why, with a relatively preserved musical thinking that allows him to hear "inside himself" a musical phrase, or even to sing a tuneful theme, to hear and to appreciate a performance, our composer cannot however express his thoughts by writing or playing them. Musical expression is in him grossly impaired. Accordingly his artistic creation is completely destroyed, as it was in the writer, and, seemingly, in a somewhat similar way, with in addition an important apraxic component, as we have already stressed. So one may see the importance of unaltered means of expression in artistic realization. To conceive is nothing, to express is all. Pleasing musical themes, and likely poetical expressions remain, doubtlessly, deeply buried in many people, who have not found, for various reasons, chiefly technical ones, the necessary means with which to express them. The cause of the failure of both our artists appears to be a technical one. Mechanisms necessary to technical accomplishment of literary or musical works have been destroyed or altered by aphasia.

These two preceding examples are in agreement with all we know about interference of language impairment upon intellectual expression during aphasia. The particular point is that our third artist escapes such a disaster in the realization of his plastic work. Let us consider the reasons. Here, the painter will draw a figurated expression, aiming to suggest an interpretation of the world, from visual perceptions, forms and

colours. That visual construction made up of drawing, composition, and colour values, evokes in the observer various reactions, ranging from direct sensory impressions, more or less marked, to various emotions in which intelligence has a part (understanding, evaluation, comparison, discussion or realization and technique) but where visual sensations are prominent (harmony, expression, climate, visual satisfaction). We know that æsthetic pleasure, before a work of art, is not merely the result of sensorial function, but, unless we discuss some modern abstract painting, we cannôt deny its main importance. Properly speaking, pictorial expression does not use a language, although sometimes the term of "pictorial language" is used. There is no plastic language, and the same is true for sculpture. There is a way of using drawing and colour to express forms and light, and an art to emphasize them. This is not the use of a language made up of symbols, it is the use of a plastic reality, more or less transposed by particular technique into a climate which, in the painter, is the exact equivalent of the language. If the climate of a Rembrandt, of a Leonardo da Vinci, or of a Turner is immediately recognizable, it is so in quite a different way than is the language of Byron or Wagner. Because his climate is a direct one, without any symbolistic medium, we are conscious of it immediately, and we simultaneously take a part in the vision he gives us. On the other hand, the figurated expression of the musician, and to some extent that of the writer, is presented to us as a whole that flows through time, made of successive phases, which we have to remember to be able to gather. Aphasia, consequently, does not alter plastic expressive power, since it takes place outside language. Only visual agnosia, an "imperception" as Jackson used to say, by altering form and colour perception would destroy their use in artistic realization. Of course, a concomitant apraxia would deprive the artist's hand of the power of laying on a canvas his personal vision. That happened in the case of our painter, fortunately only for a time. It is not without interest to notice that the most intellectual part of pictorial conception—the choice of a theme, the disposition of the subject, and the appreciation of values, i.e. everything that requires judgment—is not disturbed either. In addition to technique itself, the conceptual part of artistic realization remains unaltered by our painter's aphasia. But, as we have already mentioned, one may imagine that a painting, which is not made up of predominant sensorial components, but of abstract figuration, that is real ideograms, might be deeply altered by an aphasia. We would not, however, go so far as to hope that Picasso becomes aphasic, to show us that he can no longer paint in his present manner, but that he is still able to give us the fine paintings of his "blue period."

To summarize, we may state that the painter may continue with his

artistic production despite aphasia, because it impairs a language but leaves him able to use his visual perceptions to create, through technical means, form and light values, a sensorial use of reality, but unable to use a language made up of symbols. In agreement with Jackson's idea that aphasia does not alter the whole of intelligence, but only part of it, artistic creation becomes impossible when aphasia disturbs techniques based on language, but preserves techniques based on visual perceptions. This fact also shows that a theory, such as the German authors' "Gestalt," cannot be applied to every case, or to every form of aphasia. On the contrary it seems to agree with Henry Head's view on the part played by alteration of symbolic thinking in aphasia.

If art, as one has said, is just "overcome difficulty," one must agree that aphasia creates for writers and musicians a major, insuperable difficulty: realization of an artistic language with deteriorated tools. The painter, with an impaired language tool, may use untouched visual perceptions and continue with his artistic creation.

This implies:

(1) The different nature of the psychophysiology in artistic realization in the writer and composer on the one hand and in the painter on the other.

(2) The main part played, in the disturbance caused by aphasia, by the alteration of technique which remains localized to language.

(3) The preponderance of altered means of expression in the failure of artistic realization, as compared with preservation of conceptual thinking.

(4) Intellectual interference in aphasia, as far as artistic creation is concerned, remains localized mainly to expressive means based on language.

THE INTRANEURAL TOPOGRAPHY OF THE SCIATIC NERVE AND ITS POPLITEAL DIVISIONS IN MAN[1]

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In an earlier paper (Sunderland, 1945*a*) the intraneural anatomy of the radial, median and ulnar nerves in man was described with special reference in each case to: (1) The behaviour of the constituent funiculi and their fibre-composition; (2) the distance above the site of branching for which funiculi, representing individual muscular and cutaneous branches, retained their identity and position in the nerve trunk before participating in plexus formations; and (3) the precise nature and extent of the redistribution of the various branch fibres which resulted from the migration, division and anastomosis of funiculi. The present report is an extension of this work and covers the intraneural anatomy of the sciatic nerve and its popliteal divisions.

Material and Methods

An investigation of the blood supply of the sciatic nerve and its popliteal divisions (Sunderland, 1945*b*) involved the preparation of histological sections from segments of these nerves taken in the following regions in 40 adult dissecting-room subjects: buttock, upper, middle and distal thirds of the thigh, popliteal fossa and neck of the fibula. In other specimens the nerves were also sectioned at the mid-thigh, at the junction of its upper-fourth and lower three-fourths, and at the junction of the upper three-fourths and lower-fourth. This material proved suitable for a study of the number, size and general arrangement of the funiculi in these regions. The two divisions of the sciatic nerve were examined at corresponding levels in each subject.

For the purpose of investigating the behaviour of the funiculi at successive levels and the manner in which the branch fibres were rearranged by intraneural plexus formations, and in order to ascertain the distance above the site of branching for which funiculi, representing individual branches, retained their identity in the nerve trunk a sciatic nerve and its terminal divisions were serially-sectioned between the sciatic notch and the

[1]This work was assisted by a grant from the National Health and Medical Research Council of Australia.

tibial malleolus. The nerve and its branches were carefully and fully dissected between these levels. The branch pattern is shown in fig. 1. For purposes of identification each branch was sectioned at the site of its origin in order to establish its funicular pattern while this point was fixed with reference to a line drawn transversely across the middle of the popliteal fossa between the opposing surfaces of the femur and tibia; all measurements were made directly along the nerve. Landmarks were also established along the nerve to permit the correct identification of quadrants in serial sections. The nerve and its popliteal divisions were then divided into segments which were fixed in the usual manner, embedded in paraffin, serially-sectioned and stained with hæmatoxylin and eosin. Every tenth section was mounted and the others retained for reference. The sections were enlarged on a micro-projector, drawn and the diagrams arranged in serial fashion. The funiculus or funiculi comprising each branch were given a designatory marking and traced into and proximally along the nerve as represented by the transverse sections. In this way the position and fibre-content of a funiculus at any particular level and the manner in which its fibres were redistributed at other levels could be accurately established.

The fusion of funiculi may be followed by the complete or partial intrafunicular mixing of the fibres, which is the usual arrangement, or the elements of the two original funiculi may remain segregated in the bundle formed by the fusion. In the latter event the fibres may retain a constant position within the funiculus or move, *en bloc*, within it. The intrafunicular behaviour of fibres determines their subsequent fate when the funiculus divides and so governs the fibre-composition of the new funiculi. The rearrangement of fibres occurring within the funiculi, after the disappearance of all traces of fusion in the form of partitioning septa, could not be accurately investigated by the methods employed in the present study. For the purpose of tracing the intraneural course of peripheral branch fibres, it has, therefore, been assumed that the fusion of funiculi and the disappearance of segregating intrafunicular septa was ultimately followed by an intermingling of their constituent fibres so that when funicular division occurred the daughter bundles carried representatives of all the original fibres contained within the parent bundle. On this basis, the redistribution indicated was the maximum possible.

Transverse sections of the nerves at various levels are shown in the accompanying charts and in fig. 2; owing to the size of the nerve it was not considered practicable to represent all sections at the same magnification while the limiting ring in each diagram bears no relationship to the original limits of the epineurium. The opportunity is taken here to correct an error in the original paper (Sunderland, 1945*a*) in which the magnifica-

tion of the illustrated sections, which were to scale, was given as × 25. This was the magnification before reduction for publication—on reduction the magnification became × 9.

Additional information relating to the grosser features of the intraneural anatomy was obtained by microdissection of a further ten specimens in which the peripheral branches were traced along the nerves as far proximally as was practicable. In this connexion it is to be remembered that many of the significant funicular anastomoses are so minute that they could only be demonstrated by microscopic examination. Consequently they would not be revealed by dissection of the nerve which, if employed alone, could therefore give misleading results. In the descriptions which follow all branches have been regarded as proceeding from the structure they innervate towards, into, and along the nerve trunk so that the latter has been synthesized in terms of its branches. Similarly, in the discussion relating to the intraneural behaviour of funiculi the direction of change is from distal to proximal.

General Features

There was no exchange of bundles between the two popliteal divisions in the sciatic trunk and no difficulty was experienced in identifying and separating the two divisions macroscopically. It is to be noted, however, that the line of separation is often directed obliquely, and not directly anteroposteriorly, through the nerve so that posteriorly the medial division is overlapped by the lateral.

For convenience, the funicular pattern of each division of the dissected specimen will be considered separately.

Number and Size of the Funiculi

The bundles were more compactly arranged in some regions than in others depending upon the looseness of the supporting epineurial framework and the amount of fat which it contained. As a rule the nerve was thinner where there were few large funiculi than where the bundles were small and numerous since in the latter case the amount of supporting connective tissue was necessarily greater and naturally added considerably to the cross-sectional area of the nerve.

The anastomosis and division of funiculi were responsible for variations in the size and number of the bundles and these two features were inversely related at any level. There was no constant or characteristic pattern for either nerve. The number of funiculi varied greatly from nerve to nerve and individual to individual at any given level and at the same level on the two sides of the body. The number of funiculi observed at six levels between the sciatic notch and the neck of the fibula is shown in Table I. The number, in both divisions, diminished from the sciatic notch

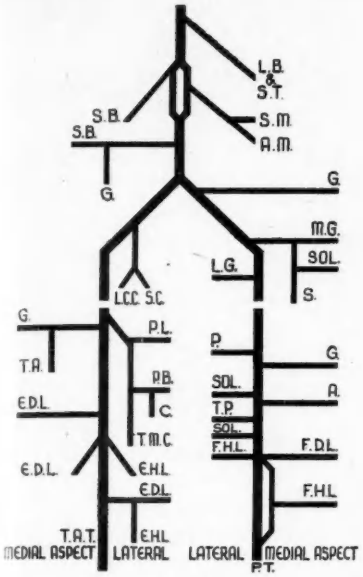
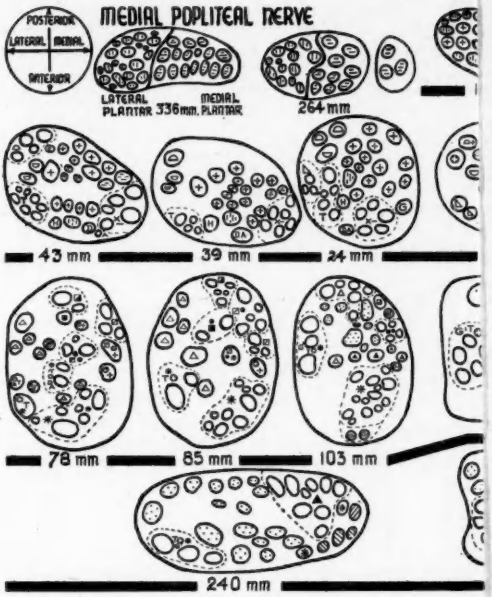


FIG. 1.



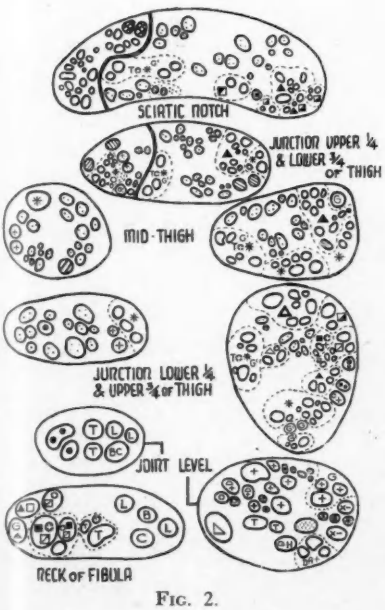
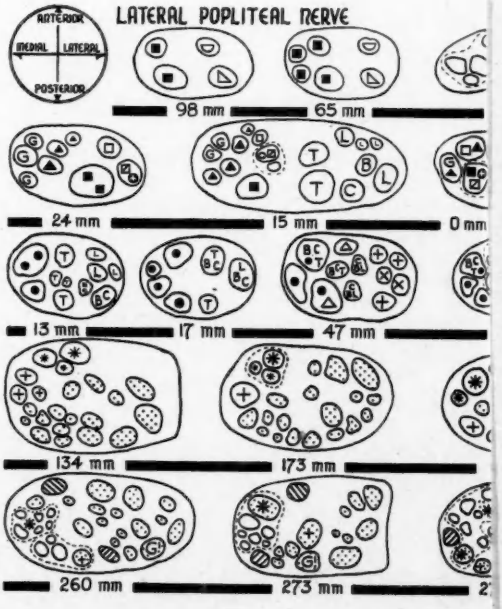
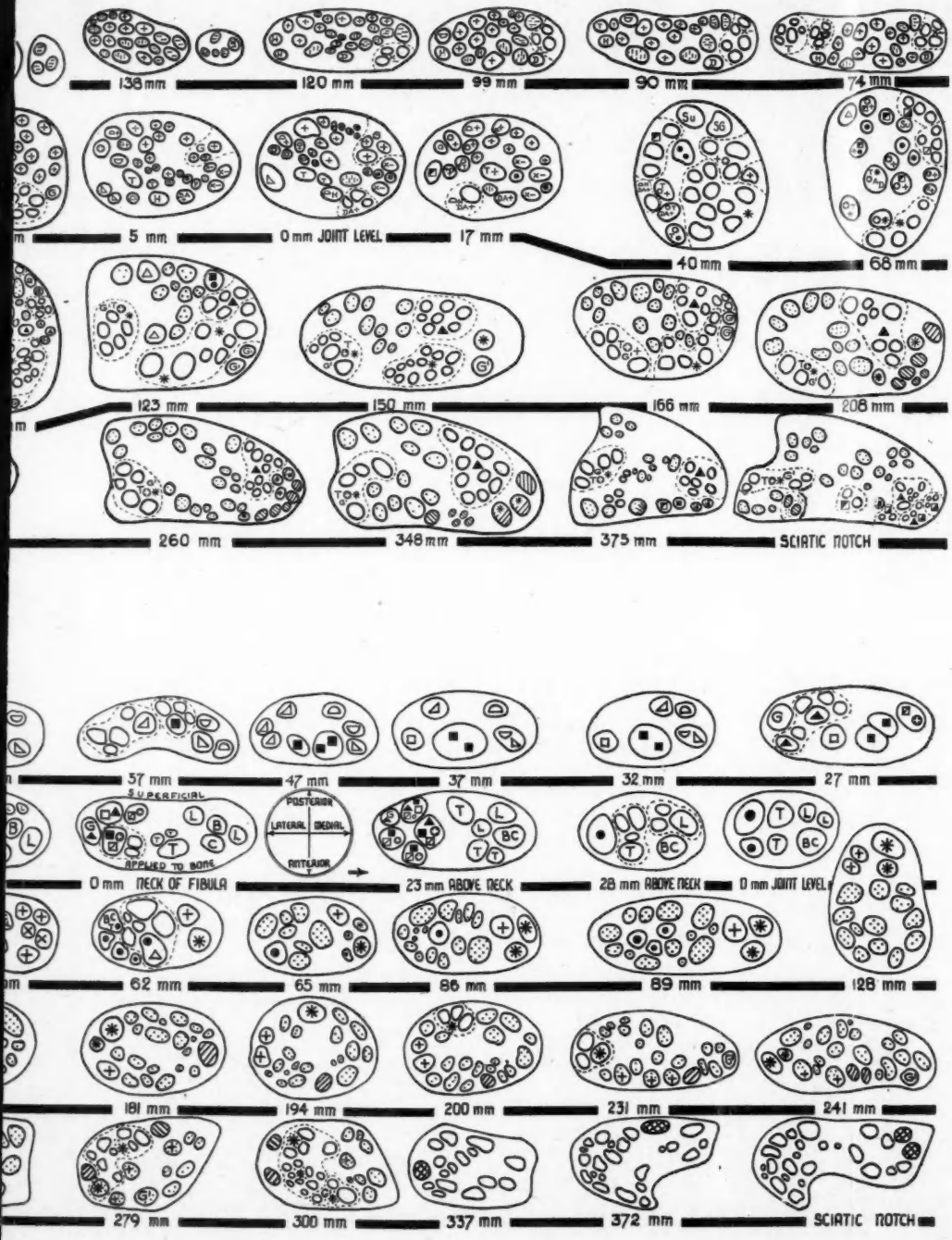


FIG. 2.



To illustrate article by S. Sunderland and L. J. Ray.


138 mm
120 mm
99 mm
90 mm
74 mm
5 mm
0 mm JOINT LEVEL
17 mm
40 mm
68 mm
123 mm
150 mm
166 mm
208 mm
260 mm
348 mm
375 mm
SCIATIC NOTCH
57 mm
47 mm
37 mm
32 mm
27 mm
SUPERFICIAL
APPLIED TO BONE
POSTERIOR
LATERAL
MEDIAL
ANTERIOR
0 mm NECK OF FIBULA
23 mm ABOVE NECK
28 mm ABOVE NECK
0 mm JOINT LEVEL
62 mm
65 mm
86 mm
89 mm
128 mm
181 mm
194 mm
200 mm
231 mm
241 mm
279 mm
300 mm
337 mm
372 mm
SCIATIC NOTCH

Plantar Nerves :

(i) Medial

(ii) Lateral

(iii) Combined fibres from both plantar nerves

Flexor hallucis longus (distal branch)

Flexor hallucis longus (distal branch) and combined plantar fibres

Flexor hallucis longus (proximal branch)

Arterial

Flexor digitorum longus

Soleus (distal branch)

Soleus (intermediate branch)

Soleus : combined fibres from the distal and intermediate branches

Tibialis posterior

Combined fibres from the plantar nerves, the tibialis posterior and the branches to the soleus (distal and intermediate)

Popliteus

Lateral head of gastrocnemius

Combined fibres from the popliteus and lateral head of gastrocnemius

Combined fibres from the soleus (proximal branch) and medial head of gastrocnemius

Sural

Combined fibres from the soleus (proximal branch), medial head of gastrocnemius and sural

Combined fibres from the popliteus, both heads of the gastrocnemius, soleus (proximal branch)

Combined fibres from the popliteus, both heads of the gastrocnemius, soleus (proximal branch) and sural

Distal genicular fibres. At and above a level 103 mm. above the joint proximal genicular fibres are associated with the sign denoting flexor hallucis longus (distal) and combined plantar fibres but they have not been included in the diagrams.

Proximal genicular fibres

Combined fibres from all branches excluding the geniculars, medial head of gastrocnemius, proximal soleus, sural and hamstrings

Combined fibres from all branches excluding the sural, flexor digitorum longus, arterial and hamstrings

Combined fibres from all branches excluding the hamstrings

At and above a level 208 mm. above the joint :

Adductor magnus

Semimembranosus

Long head of biceps and semitendinosus

Combined fibres from all three hamstrings, plantars, flexor hallucis longus

Combined fibres from adductor magnus, semitendinosus, long head of biceps, plantars, flexor hallucis longus

Combined fibres from adductor magnus, semimembranosus, flexor hallucis longus, plantars

Anterior Tibial Nerve :

Terminal section of the anterior tibial nerve

Extensor hallucis longus (distal branch)

Extensor hallucis longus (proximal branch)

Combined extensor hallucis longus fibres

Extensor digitorum longus (distal branch)

Extensor digitorum longus (intermediate branch)

Combined fibres from the distal and intermediate branches to the extensor digitorum longus

Combined fibres from the proximal and intermediate branches to the extensor digitorum longus

Tibialis anterior

Distal genicular fibres

Combination of all fibres

Musculocutaneous Nerve : In the diagrams this division has not been represented below a point 15 mm. below the fibular neck.

Terminal section of the musculocutaneous nerve

Peroneus brevis

Cutaneous

Peroneus longus

Combination of all fibres

Lateral Popliteal Nerve :

Combination of all fibres from the anterior tibial and musculocutaneous divisions

Sural communicating

Lateral cutaneous of the calf

Combined sural communicating and lateral cutaneous of the calf

Short head of biceps (distal branch)

Short head of biceps (proximal branch)

Combined fibres from the short head of the biceps

Proximal genicular fibres

Above a level 300 mm. above the joint the funiculi without a marking contain fibres from every branch of the nerve excluding the geniculars fibres (which are contained only in bundles located anterolaterally) and the fibres from the short head of the biceps.

TABLE I.—THE NUMBER OF FUNICULI AT VARIOUS LEVELS IN THE POPLITEAL DIVISIONS OF THE SCIATIC NERVE

Specimen	*Sciatic notch*	*Junction of the upper fourth and lower three-fourths of the thigh*	*Mid-thigh*	*Junction of the upper three-fourths and lower fourth of the thigh*	*Mid-popliteal*	*neck of the fibula*
(1) Medial	137	83	52	36	32	48
Lateral		31	23	12	6	14
(2) Medial	54	25	27	17	20	21
Lateral		26	6	4	4	21
(3) Medial	51	27	13	11	16	20
Lateral		18	20	4	5	10
(4) Medial	42	25	27	17	23	19
Lateral		15	13	1	6	11
(5) Medial	67	38	32	20	36	26
Lateral		15	11	8	15	24
(6) Medial	43	33	31	25	16	23
Lateral		9	12	9	1	14
(7) Medial	136	93	59	47	54	28
Lateral		36	34	7	27	17
(8) Medial	65	32	36	28	25	22
Lateral		16	11	8	6	21
(9) Medial	92	41	32	30	16	24
Lateral		21	15	4	1	16
(10) Medial	43	38	41	56	33	37
Lateral	25 (68)	22	22	18	7	13

down the thigh and then suddenly increased again at the knee where, however, the number did not approach that observed in the upper thigh and buttock. In general, the largest funiculi were observed in the medial popliteal division though the very largest seen occurred in those cases in which the lateral popliteal nerve was composed of a single bundle in the distal part of the thigh.

Cross-Sectional Area of Individual Branch Fibre Systems Within the Nerve

Though the cross-sectional area of a funiculus is not an accurate measure of its fibre content, since the degree of myelination, the amount of the intrafunicular connective tissues and the amount of shrinkage occurring during preparation are contributing factors, it may nevertheless be accepted as an indication of the proportionate area occupied by its constituent fibres in the entire nerve. The cross-sectional area of the funiculi devoted to individual muscular and cutaneous branches before interfunicular mixing occurred has been estimated by means of a planimeter in specimens of the radial, median, ulnar and sciatic nerves. The results of this

investigation are to be reported separately (Sunderland and Bedbrook, 1948) but the average values for the branches of ten sciatic nerves are given in Table II.

TABLE II.—THE PERCENTAGE CROSS-SECTIONAL AREA, AT THE SCIATIC-NOTCH OF THE FUNICULAR SURFACE OF EACH POPLITEAL NERVE OCCUPIED BY THE FIBRES OF INDIVIDUAL BRANCHES. THE VALUES GIVEN ARE THE MEANS OF TEN READINGS

Lateral popliteal division		*Medial popliteal division*	
Biceps femoris	7	Semimembranosus	4
Peroneus longus	9	Semitendinosus	4
Peroneus brevis	5	Biceps femoris	6
Total peronei	14	Total hamstrings	14
Tibialis anterior	8	Adductor magnus	4
Extensor digitorum longus	5	Medial head of gastrocnemius	5
Extensor hallucis longus	3	Lateral head of gastrocnemius	3
Extensor digitorum brevis	1	Soleus	8
Sural communicating	9	Total calf muscles	16
Lateral cutaneous of the calf	11	Popliteus	3
Cutaneous of the musculocutaneous nerve	25	Tibialis posterior	3
Terminal sensory branch of the anterior tibial nerve (includes cutaneous, articular and arterial fibres)	9	Flexor digitorum longus	2
		Flexor hallucis longus	2
		Sural	5
		Medial calcaneal	6
Arterial	2	Arterial	1
Articular	6	Articular	3
Total muscular	38	Plantars	41
Total sensory (cutaneous and articular)	60	Total muscular	44
		Total sensory (cutaneous and articular)	14
Total arterial	2	Total plantars	41
		Total arterial	1

Fibre Composition of Funiculi

Funiculi were composed of (i) muscular fibres from a single muscle, from a group of muscles having the same or similar actions, or from a number of muscles which were not functionally related; (ii) cutaneous fibres; (iii) arterial fibres; (iv) articular fibres, and (v) varying combinations of these.

The manner in which the fibres were combined in a funiculus depended on the level at which an analysis was made. At the buttock it was usual for the majority of the funiculi to contain representative fibres from most, if not all, of the peripheral branches. At lower levels a regrouping of the fibres was gradually effected so that they came to be arranged in the funiculi in various combinations with, however, a progressively diminishing number of fibre types in combination in each funiculus. A further extension of this process ultimately resulted in the appearance of groups of funiculi in which the nerve fibres were destined solely for specific branches.

The Behaviour of Funiculi

The funiculi were subjected throughout the length of both nerves to intraneural migrations, anastomoses and divisions.

The intraneural shift or migration of funiculi.—On occasion some bundles did not pursue a straight course in the nerve but ascended obliquely through it or over its surface. Thus, where a muscle was innervated by multiple branches entering different aspects of the nerve at different levels, the bundle or bundle group representing the branches sometimes migrated considerable distances in this way in order to reach, and anastomose with each other. An illustration is provided by the extensive migration undertaken by the bundle representing the distal branch to the short head of biceps to reach and anastomose with that from the proximal branch.

There was also a tendency for funiculi from different muscles having similar or associated actions or from associated cutaneous areas to anastomose with one another before participating in further communications. This sometimes led to a considerable intraneural shift of one or other or both bundle systems before a union was effected, and in this way the constituent fibres of the funiculi were transported, *en masse*, from one quadrant to another. An illustration is provided by the behaviour of the fibres from the soleus and gastrocnemii.

Sometimes, however, an alteration of shape of the nerve induced a redistribution of the fibre systems with reference to their quadrantic localization, though they retained the same relationship to one another—such an example was provided in the case of the medial popliteal nerve as it approached the mid-third of the thigh from the popliteal fossa.

The anastomosis and division of funiculi.—The plexus formations were of three fundamental types:

(1) The first occurred between bundles whose constituent fibres were derived exclusively from one branch and were consequently localized to individual bundle groups. The anastomoses involved adjacent bundles in the group but not all at the same level. The scattering and intermingling of fibres thereby effected involved fibres from the same source so that these remained confined within the limits of the bundle group. As a result the degree of localization obtaining was sharp.

(2) The members of one bundle group anastomosed with the adjacent bundles of another and this was followed by a blending of the fibres of the two originally distinct groups. The intermingling was effected gradually since the anastomoses involved only immediately adjacent bundles. Bundle groups from different muscles having similar actions or from associated cutaneous areas often participated in this type of plexus formation in the initial stages of their ascent along the nerve (e.g. gastrocnemius

and soleus fibres). Thus fibres could only be widely dispersed by slow transference through a number of bundles so that complete mixing occurred only after several centimetres. Moreover even when the intermingling was completed, unless individual migrations had increased the range of distribution, the fibres of the two systems were still confined to that portion of the nerve originally occupied by the two bundle systems. In this process one bundle group may proceed without change of position while the fibres of another are progressively added to it. Thus in addition to promoting the intermingling of fibres from different sources, this type of intercommunication may lead not to an exchange of fibres but to the absorption of the constituent fibres of one bundle group into another.

(3) The third type of intercommunication was observed at proximal levels and was responsible for the most widespread dispersal of the fibres. Here, neighbouring bundles composed of a combination of different branch fibres, which had intermingled at distal levels, repeatedly divided and anastomosed. The fibres were in this way progressively but gradually diffused over the funiculi until considerable scattering had been effected.

Funicular intercommunications took place along the entire course of the nerve. The maximum length of nerve trunk seen with a constant pattern was 6 mm. though individual bundles and bundle groups pursued longer courses. The intercommunications were of such a nature that the dispersal of fibres which they occasioned was gradual and, at any one level, produced only a minor degree of intraneural shift. At the same time it is to be noted that the continuous exchange of fibres occurring among the various funiculi as the result of their fusion and division can only mean a continuous modification of the disposition and arrangement of the fibres, relative to one another, throughout the length of the nerve. It was, however, the summation of the changes at successive levels, and not that at any one level, which was ultimately responsible for the widespread scattering of the fibres. At proximal levels the constituent fibres of the bundles were arranged in varying combinations and proportions though no single bundle necessarily contained fibres from every branch. It is of interest that the intercommunications did not invariably lead to a wide dispersal of the fibres but that in a very few cases the process was actually reversed so that fibres, which had been widely scattered over several funiculi at distal levels, were regathered into one or two bundles. Thus all the fibres of the anterior tibial nerve were collected into a single bundle 26 mm. above the neck of the fibula. The funicular distribution of the branch fibres at different levels is shown in the diagrams of the transverse sections illustrating the intraneural topography of the popliteal nerves.

Thus despite the changing plexiform character of the funicular pattern, fibres from peripheral branches pursued a localized course in the nerve

for variable, though often considerable, distances. Such a localization involved all or the majority of the fibres of any branch. It is evident, therefore, that the redistribution implied in the plexus principle is, in the first instance, local and not general.

The Lateral Popliteal Nerve Below the Level of the Knee-Joint

Musculocutaneous Division

The terminal section of the nerve.—The bundle group representing the terminal section of the nerve proceeded to the joint level as an independent system of 1 to 6 funiculi.

Branch furnishing a twig to the peroneus brevis muscle and another to the skin.—The common stem of origin branched from the musculocutaneous nerve 62 mm. below the neck of the fibula. Each twig was composed of a single bundle which proceeded along the trunk for 82 mm. and then fused to form a compound funiculus which continued as such to the joint level.

Branch to the peroneus longus.—The bundle group representing this branch joined the musculocutaneous trunk 15 mm. below the neck of the fibula and continued as a separate group to the joint level.

Anterior Tibial Division

The terminal section of the anterior tibial trunk.—The terminal anterior tibial fibres (from the foot) proceeded as a separate bundle group to a point 12 mm. below the fibular neck. At this level one of the two bundles comprising the group was joined by a bundle containing fibres from the second and third digital extensor systems. 2 mm. further proximally the second funiculus divided and each division soon received fibres from the second and third digital extensor systems; one received the extensor fibres just below the neck of the fibula and the other at that level. In the lower part of the leg the bundles varied in number from 4 to 6 and in the upper part of the leg, 1 to 4.

Branch to the tibialis anterior and knee-joint.—The twigs to the tibialis anterior and knee-joint shared a common stem of origin which branched from the anterior tibial nerve 28 mm. below the neck of the fibula.

The tibialis anterior bundle group proceeded to a level 10 mm. below the neck of the fibula, where one of the three bundles fused with a funiculus containing fibres from the first and second digital extensor systems. At the neck of the fibula the second tibialis anterior bundle joined this compound funiculus while the third joined a funiculus composed of articular fibres, and this, after a course of 25 mm., united with a bundle composed of fibres collected from all the other branches.

Articular fibres joined the anterior tibial trunk as a single funiculus. This system proceeded, with funicular divisions and reunions, to the neck

of the fibula where the bundle of fibres at that level was joined by one composed of tibialis anterior fibres—the subsequent fate of this compound funiculus has been considered.

Branches to the extensor digitorum longus and extensor hallucis longus.—There were three sets of branches to the two toe extensors; the proximal supplied the extensor digitorum longus, in the second the branches to the two muscles arose separately at the same level; while the third or distal divided to supply both muscles. For convenience and to avoid confusion these have been referred to as the first, second and third digital extensor systems.

(i) Third digital extensor system (the distal extensor digitorum longus and the distal extensor hallucis longus): The common stem of origin of the two branches joined the anterior tibial trunk 103 mm. below the neck of the fibula. The fibres of each were contained in a single funiculus. These proceeded centrally for 57 mm., fused, and the compound funiculus continued for a further 15 mm. when it was joined by two bundles from the second digital extensor system: one of these contained all the extensor hallucis fibres and the other a proportion of the extensor digitorum fibres of that system. The compound funiculus continued to a point 18 mm. below the fibular neck where it bifurcated: one division united, 6 mm. further proximally, with a bundle containing fibres from the terminal section of the anterior tibial nerve, while the second continued to a point above the fibular neck before fusing with a bundle containing fibres from the digital musculature and tibialis anterior.

(ii) Second digital extensor system: The intermediate branch to the extensor digitorum longus and the proximal branch to the extensor hallucis longus joined the anterior tibial nerve 61 mm. below the neck of the fibula.

The extensor digitorum longus fibres formed a bundle group which was reduced to 2 bundles, 44 mm. below the neck of the fibula. At this level one bundle fused with another composed of fibres from the first digital extensor system while the other migrated obliquely over the surface of the nerve to reach and anastomose with (31 mm. below the neck) the funiculus containing all the fibres from the third, and the hallucis fibres from the second, digital extensor systems.

The extensor hallucis longus fibres were contained in a single funiculus which continued as such till it fused, 31 mm. below the fibular neck, with the funiculus containing the fibres from the third digital extensor system.

(iii) First or proximal digital extensor system: The fibres were from the extensor digitorum longus and were contained in three bundles which joined the anterior tibial trunk 45 mm. below the neck of the fibula. Within a millimetre the bundles had fused and the funiculus thereby

formed anastomosed with a bundle containing extensor digitorum fibres from the second digital extensor system. This compound funiculus was later joined by two bundles containing fibres from the tibialis anterior; one joined 10 mm. below the neck of the fibula and the other at the level of the neck.

Localization

The position occupied by the different branch fibres as they were traced along the anterior tibial and musculocutaneous divisions is illustrated in the chart of the lateral popliteal nerve sections. The number and arrangement of the funiculi, their composition, and the distribution of the fibres in each division at the neck of the fibula and at the joint level are also illustrated in fig. 2. Between these two levels the maximum length for which each division retained a constant funicular pattern was 3 mm. The fibres of all branches of the anterior tibial nerve were contained in a single bundle 26 mm. above the neck of the fibula and in two bundles at the joint level.

The Lateral Popliteal Nerve Between the Sciatic Notch and the Knee-joint

It is to be noted that, as the nerve turns from the neck of the fibula to ascend in the popliteal region, the surface of the nerve originally applied to the bone becomes anterior and the superficial surface posterior, while the anterosuperior border becomes lateral.

Musculocutaneous Nerve

The terminal section of the nerve.—The bundle group (2 to 4 funiculi) representing the terminal section of the nerve continued for 16 mm. One of the two component bundles then joined another containing fibres from the peroneus brevis and the associated cutaneous branch while a derivative of this compound funiculus received the second bundle 2 mm. further proximally.

The terminal fibres at first occupied the lateral section of the musculocutaneous trunk, that is, the central third of the lateral popliteal nerve, the bundles extending to the anterior and the posterior surface. Recurrent anastomoses with the bundles of neighbouring groups, which commenced 16 mm. above the joint, resulted in their gradual dispersal. However, they were still relatively concentrated centrally, in combination with others, at a level 62 mm. above the joint, owing to the fact that the sural communicating and lateral cutaneous of the calf bundle groups had joined the medial aspect of the lateral popliteal nerve. Proximal to this level the terminal fibres were slowly scattered over the remaining funiculi comprising the nerve with the exception of those devoted to the sural communicat-

ing and lateral cutaneous of the calf which were only involved at very high levels.

The peroneus brevis and associated cutaneous fibres.—The bundle containing these fibres divided into two, 12 mm. above the joint level. 4 mm. further proximally one division anastomosed with a bundle carrying peroneus longus fibres and the other with a bundle containing terminal fibres. 19 mm. above the joint all bundles in the musculocutaneous division of the lateral popliteal nerve contained fibres from the peroneus brevis and the associated cutaneous branch.

The fibres were placed in the anteromedial quadrant for 16 mm. They then commenced to scatter laterally towards the centre and posteriorly over the medial aspect of the nerve though they remained concentrated medially until the bundle group representing the sural communicating and lateral cutaneous nerve of the calf joined the medial aspect of the nerve, when the brevis and associated cutaneous fibres naturally became central. All traces of localization were lost, 61 mm. above the joint level, by further scattering laterally.

The peroneus longus.—The bundle group carrying the peroneus longus fibres ascended in the posteromedial quadrant for 16 mm., when one of the two component bundles united with another bundle containing peroneus brevis and cutaneous fibres; 1 mm. higher this compound funiculus was joined by the remaining peroneus longus bundle. These fibres were then gradually distributed over the medial half of the nerve but were later forced centrally by the entrance, medially, of the bundle group representing the sural communicating and lateral cutaneous of the calf. The peroneus longus fibres then spread laterally until, 64 mm. above the joint, every funiculus, excluding the sural communicating and lateral cutaneous members and one composed solely of anterior tibial division fibres, contained peroneus longus fibres in combination with representatives from every other branch below the knee.

Anterior Tibial and Musculocutaneous Divisions of the Lateral Popliteal Nerve

The two divisions remained separate for 22 mm. when one of four anterior tibial funiculi was joined by one from the musculocutaneous division containing all but peroneus longus fibres. Above this level the recurrent division and anastomosis of bundles resulted in the mixing of fibres of both divisions until, 100 mm. above the joint, all the bundles contained fibres from all the branches of both divisions. 31 mm. above the joint one bundle remained which contained only anterior tibial division fibres and this was located on the extreme lateral surface of the nerve. This bundle occupied this position for 21 mm. when it divided; the subdivisions migrated internally to blend with other bundles though one

retained its identity and ascended in the centre of the nerve before finally dividing and anastomosing with other bundles 100 mm. above the joint.

The anterior tibial fibres occupied the lateral half of the nerve for 51 mm.; some bundles containing only anterior tibial division fibres were still situated anterolaterally as far proximally as a point 75 mm. above the joint.

The musculocutaneous fibres occupied the medial half of the nerve until the bundle groups representing the sural communicating and the lateral cutaneous of the calf joined the medial surface of the nerve 22 mm. above the joint, and forced them centrally. Above this level the musculocutaneous fibres remained relatively concentrated centrally for a further 29 mm.

Thus the three bundles groups comprising the musculocutaneous division of the lateral popliteal nerve (terminal musculocutaneous, peroneus longus and peroneus brevis with cutaneous) commenced to anastomose 16 mm. above the joint while the funiculi of the anterior tibial and musculocutaneous groups commenced to mix 6 mm. further proximally.

Sural Communicating Nerve and the Lateral Cutaneous Nerve of the Calf

The fibres of these two cutaneous branches shared a common stem of origin which branched from the nerve 22 mm. above the joint. The lateral cutaneous and sural communicating bundle groups each retained its identity for 37 mm. After anastomosis the combined system proceeded as a discrete unit for a further 144 mm. when interlacing commenced with funiculi containing other fibres.

Lateral cutaneous nerve of the calf.—The fibres of this branch were contained in a single bundle which divided after a course of 15 mm. The two divisions reunited 16 mm. further proximally and, 6 mm. higher, fused with a bundle composed of sural communicating fibres. From this level onwards the fibres were associated with those of the sural communicating branch and will be described with them.

Sural communicating nerve.—The bundle group representing these fibres proceeded for 37 mm. when one of the two bundles representing the group received the lateral cutaneous fibres; the other continued (1 or 2 funiculi) for a further 144 mm. before exchanging fibres with a funiculus composed of anterior tibial and musculocutaneous fibres. The complex formed by the fusion of sural communicating and lateral cutaneous fibres continued for 210 mm. when one of the two bundles comprising the group at that level fused with another composed of anterior tibial and musculocutaneous fibres, while within 2 mm. the other had joined a bundle containing anterior tibial, musculocutaneous and sural fibres.

The sural communicating and lateral cutaneous bundle group joined

and occupied the medial aspect of the lateral popliteal nerve for 87 mm. when it turned on to the posterior surface of the nerve and continued thus for 22 mm. It then passed to the posterolateral aspect and finally to the lateral margin of the nerve which it reached 176 mm. above the joint. It remained in this position for 27 mm. during which it was invaginated by bundles from the interior of the nerve which resulted in the splitting of the bundle group into two divisions. The fibres occupying the anterolateral quadrant continued in this position for 25 mm. when they were dispersed, in combination with anterior tibial and musculocutaneous fibres, anteromedially around the surface. The fibres occupying the posterolateral quadrant continued in this position (ultimately mixing with those from the anterior tibial and musculocutaneous divisions) for 92 mm. (295 mm. above the joint) when the fibres commenced to scatter medially. Within a few millimetres the sural communicating and lateral cutaneous nerve fibres were widely distributed over the lateral popliteal division and had reached its medial surface.

Genicular Branch and the Distal Branch to the Short Head of Biceps

These two branches shared a common stem of origin which branched from the anterior surface of the sciatic nerve 150 mm. above the joint. The fibres of each branch were contained in a single bundle which ascended independently, anteriorly between the two popliteal divisions. The biceps and genicular fibres could be considered as being incorporated within the lateral popliteal nerve after courses of 30 and 77 mm. respectively.

Genicular branch.—After its incorporation in the nerve the genicular bundle proceeded obliquely across its anterior face (receiving after a course of 17 mm. a bundle containing anterior tibial and musculocutaneous fibres) and reached the mid-point of that surface 273 mm. above the joint. The compound funiculus ascended in this position for 19 mm. before anastomosing with a bundle carrying fibres from all the more distal branches. 7 mm. further proximally this compound bundle split into two funiculi which continued in a mid-anterior position for 28 mm. before turning towards the lateral margin of the nerve which they reached after a course of 10 mm. (337 mm. above the joint). This lateral position was retained for 23 mm. but above this point the bundles were involved in further anastomoses. The genicular fibres, however, remained concentrated in the anterolateral quadrant of the nerve at the sciatic notch.

Distal branch to the short head of biceps.—The fibres were contained in a single bundle which split, after an intraneural course of 60 mm., into two divisions. These reunited 10 mm. further proximally and the single bundle continued as such for 87 mm. when it fused with that representing the proximal branch to the short head of biceps with which it will be

described. The fibres occupied the anteromedial aspect of the lateral popliteal division of the sciatic nerve for 8 mm. (to a point 188 mm. above the joint) and then migrated laterally across the anterior face of the nerve to reach the extreme lateral margin 275 mm. above the joint. They continued along this margin for 62 mm. before uniting with the bundle from the proximal branch to the short head of biceps.

Proximal Branch to the Short Head of Biceps

The bundle containing the fibres of this branch joined the posterolateral aspect of the nerve 260 mm. above the joint. It ascended in this position for 31 mm. when it migrated towards the lateral margin of the nerve which it reached 297 mm. above the joint. It occupied this position for 40 mm. when it fused with the bundle representing the distal branch. The bundle, which now contained all the fibres from the short head of the biceps, migrated medially over the posterior surface. The middle of that surface was reached 353 mm. above the joint and the posteromedial angle of the lateral popliteal a few millimetres below the sciatic notch. The bundle divided before reaching the notch but the two divisions remained discretely localized.

The extensive migration undertaken by the bundles from the two branches to the short head of biceps in order to reach and fuse with one another was a striking feature of the funicular pattern.

Constancy of the Lateral Popliteal Funicular Pattern

The maximum length of nerve with an absolutely constant funicular pattern was 6 mm.

Localization in the Lateral Popliteal Nerve

The localization of branch fibres within the nerve at the sciatic notch, mid-thigh, at the junction of the upper fourth and lower three-fourths and upper three-fourths and lower fourth of the thigh and at the neck of the fibula is shown in fig. 2. The intraneural topography at other levels is illustrated in the accompanying chart.

The Medial Popliteal Nerve from the Tibial Malleolus to the Sciatic Notch

Attention is directed to a change in shape of the nerve as it passed from the popliteal fossa towards the mid-third of the thigh; the appearance in transverse section altered from circular to oval with the long axis directed anteroposteriorly. This change of shape resulted in a redistribution of all fibre systems with reference to their quadrantic localization. The movement induced was in a clockwise direction (observed from below) and rotated the intraneural contents, *en masse*, through approximately 45 degrees.

Plantar Nerves

The medial and lateral plantar bundle groups remained separate for 97 mm. when fibre mixing commenced in the region of the mid-anterior surface of the posterior tibial nerve. Further intermingling took place slowly and it was not until 24 mm. above the knee-joint that the fibres of the medial and lateral plantar nerves were combined in all the bundles in which plantar fibres were represented.

Medial plantar group.—Some of the medial plantar funiculi separated from the medial aspect of the posterior tibial nerve 21 mm. above its distal end and ascended parallel to the main trunk. After a course of 105 mm. the offshoot was joined by a bundle carrying flexor hallucis longus fibres (distal branch) which entered one of the medial plantar funiculi 54 mm. further proximally. 135 mm. below the joint level all fibres in the offshoot (plantar and flexor hallucis) were contained in a single bundle which rejoined the posterior tibial nerve within 5 mm.

The medial plantar fibres in the main trunk were placed medially or anteromedially for 305 mm. when the last bundle composed exclusively of these fibres was eliminated. However, bundles from the offshoot composed of medial plantar and flexor hallucis fibres remained medially or anteromedially for a further 36 mm. (just below the joint) when lateral plantar fibres commenced to invade them; the intermingling of all three was completed 24 mm. above the joint.

Lateral plantar group.—The lateral plantar group was situated laterally for 153 mm. when superficial extensions in a medial direction were present anteriorly and posteriorly. Within 20 mm. the system had separated into antero- and postero-lateral groups. The former slowly migrated anteriorly and the latter, laterally; both were gradually reduced by anastomosis with medial and combined plantar funiculi until, at a level 136 mm. below the joint, a small group of lateral plantar funiculi persisted anteriorly. The group (3 to 6 funiculi) then commenced to move deeply into the nerve but within 50 mm. the bundles were again concentrated anteriorly. They remained in this position being slowly reduced by the passage of fibres to the adjoining combined plantar funiculi until only one bundle remained. This continued anteriorly until it was displaced centrally by the migration of the funiculi representing other fibres. It then ascended in a central position until it was eliminated by anastomosis with one containing other fibres, 24 mm. above the joint.

Localization of the combined medial and lateral plantar funiculi and fibres.—98 mm. above the distal extremity of the nerve the funiculi composed of fibres from both plantar nerves were located posteriorly and centrally. 75 mm. further proximally plantar fibres were contained in funiculi at the lateral margin of the nerve, though the concentration was

still predominantly central. 30 mm. higher, the fibres were distributed posterolaterally. They then infiltrated medially across the posterior surface but, as other branches were added to the nerve at more proximal levels, they came to occupy the central two-fourths of the nerve and were then gradually infiltrated by the fibres of other branches. The last bundle composed solely of medial and lateral plantar fibres terminated 42 mm. above the joint.

At the sciatic notch the plantar fibres were represented in all funiculi; in some, anteromedially, they were associated with fibres from the hamstrings, flexor hallucis longus and the knee-joint while in two small bundle groups located laterally in the medial popliteal division they were associated with fibres from the flexor hallucis longus, tibialis posterior, proximal genicular and distal and intermediate soleus branches. Elsewhere they were intermingled with fibres from all or most of the branches of the medial popliteal nerve except those to the hamstrings.

Flexor Hallucis Longus

This muscle was supplied by two branches. The distal branch joined the medial plantar offshoot of the posterior tibial nerve as a single funiculus which, after a course of 54 mm. anastomosed with one of the medial plantar group; the subsequent fate of its fibres has been described with that group.

The fibres of the proximal branch joined the anterolateral aspect of the posterior tibial nerve 132 mm. below the joint and remained in this position for 84 mm.; the bundle group was situated just anterior to the funiculus from the distal branch to the soleus while the bundle group from the tibialis posterior entered on its lateral side. The group then drifted medially across the anterior face of the nerve and within 9 mm. had reached the mid-anterior surface along which it ascended for 38 mm. before fusing with one of the funiculi containing fibres from the intermediate branch to the soleus. The compound bundle thereby formed continued up the mid-anterior surface for a further 17 mm. before anastomosing with a funiculus composed of fibres from both plantar nerves, the arterial branch and the branch to flexor digitorum longus.

Above the joint the flexor hallucis longus fibres (from both branches) were at first placed in the anteromedial quadrant but 30 mm. above the joint they were distributed over the entire anterior half of the nerve; 26 mm. higher they were infiltrating into the posterolateral quadrant while 44 mm. further proximally they were widely distributed through the nerve except at the extreme posteromedial angle. Above this level they were scattered over the remaining bundles.

Flexor Digitorum Longus

The branch entered the nerve 132 mm. below the joint, as a single bundle. After a course of 93 mm. this was joined by the arterial funiculus and 24 mm. further proximally it received a bundle composed of fibres from both plantar nerves. The compound bundle divided at the joint level; one division rapidly migrated and joined a bundle containing intermediate soleus and flexor hallucis fibres while the remaining fibres continued as a group of one to two funiculi for a further 35 mm. before blending with fibres from the plantar, tibialis posterior and intermediate and distal soleus branches.

The flexor digitorum bundle joined the anterior aspect of the nerve and gradually drifted medially until, after a course of 30 mm., it had reached the medial margin of the nerve. The fibres were then placed in an anteromedial or mid-anterior position for 119 mm., first as a discrete system and later in combination with other fibres. This system then migrated laterally and 23 mm. further proximally (40 mm. above the joint) the flexor digitorum fibres were located in the anterolateral quadrant in association with arterial, plantar, flexor hallucis, soleus and tibialis posterior fibres. This position was retained for 16 mm. when further migration scattered the fibres centrally and into the posterolateral quadrant where they were confined at a level 81 mm. above the joint, but 114 mm. further proximally some fibres had infiltrated to the anterior surface of the nerve. This infiltration continued until, at the sciatic notch, the flexor digitorum fibres were widely distributed throughout the medial popliteal division with the exception of its anteromedial corner and a small bundle group located laterally.

Arterial Branch

A funiculus composed of arterial fibres joined the anteromedial aspect of the nerve 57 mm. below the joint. After a course of 18 mm., during which it migrated laterally, the bundle anastomosed on the mid-anterior face of the nerve with the flexor digitorum funiculus. The subsequent behaviour of the arterial fibres has been described with those of the flexor digitorum longus.

Tibialis Posterior

The fibres comprising this branch joined the lateral margin of the nerve 90 mm. below the joint. The bundle group was placed at the extreme lateral edge of the nerve for 46 mm. when it drifted across the anterior face of the nerve as it ascended. 10 mm. above the joint, the largest of three bundles joined a funiculus composed of fibres from the distal and intermediate branches to the soleus and from this point proximally was associated with this system. The remaining two bundles fused,

4 mm. further proximally, with a bundle composed of plantar fibres and, after a course of 8 mm., this anastomosed with another bundle composed of intermediate soleus and tibialis posterior fibres. The tibialis posterior fibres, in association with those from the soleus and plantar branches, then moved into the anterolateral quadrant and, fanning out, were distributed laterally and centrally at a level 31 mm. above the joint; 7 mm. higher they had a predominant localization in the lateral half of the nerve. Infiltration in a medial direction then occurred and, as a result, fibres reached the medial margin of the nerve 85 mm. above the joint. Further scattering of fibres carried the tibialis posterior representatives into the anteromedial quadrant but at the sciatic notch there were bundles located anteromedially which did not contain them. At the notch there was a small bundle group laterally (i.e. central part of the sciatic nerve) in which the tibialis posterior fibres were in combination with fibres from the plantar nerves, flexor hallucis longus, the distal and intermediate soleus, and the proximal genicular branches—elsewhere they were in combination with those from all branches distal to the hamstrings.

Popliteus and Lateral Head of Gastrocnemius

Though the branches to these two muscles entered the nerve at different levels, the bundles representing them soon anastomosed. The two systems may, therefore, be conveniently considered together.

The branch to the popliteus joined the extreme lateral margin of the nerve 30 mm. below the joint. The bundle group occupied this position for 5 mm. and then became anterolateral in front of the group representing the distal and intermediate fibres to the soleus. It continued thus and was approached laterally, 3 mm. above the joint, by the bundle of the lateral head of the gastrocnemius which entered the nerve at that level. The two bundle groups coursed upwards for 10 mm. and then commenced to anastomose. Within a millimetre the fibres from the two systems were mixed and contained in two bundles which were located laterally. The complex was placed laterally (1 to 3 bundles) for 24 mm. when the bundles commenced to migrate posteriorly towards those devoted to the sural, medial head of gastrocnemius and proximal soleus fibres. 48 mm. above the joint the popliteus and lateral gastrocnemius fibres were contained in a few bundles which were widely scattered over the posterolateral quadrant. One of the bundles then fused with another containing fibres from the plantar nerves, the flexor digitorum and flexor hallucis, arterial, tibialis posterior and distal and intermediate branches to soleus. Above this level there was a slow infiltration of the popliteus and lateral gastrocnemius fibres posteriorly where they intermingled with those, recently added, from proximal genicular and the distal and intermediate branches to the soleus,

*The medial head of gastrocnemius and the proximal branch to soleus.

with which they were subsequently associated. They were slowly scattered over the posterior half of the nerve and 92 mm. above the joint were present in all bundles except those located anteriorly. At the sciatic notch they were distributed over the entire medial popliteal division of the sciatic nerve with the exception of: (i) some funiculi situated anteromedially which contained only fibres from the hamstrings, plantar nerves and flexor hallucis longus, and (ii) one small group laterally which contained only fibres from the plantar nerves, tibialis posterior, flexor hallucis longus, the proximal genicular and the distal and intermediate branches to the soleus.

Soleus

The soleus was innervated by proximal, intermediate and distal branches. The proximal shared a common stem of origin with the branch to the medial head of the gastrocnemius which will be described with it.

Distal branch.—The fibres of this branch joined the nerve 111 mm. below the joint and coursed for 100 mm. when a small bundle anastomosed with one containing fibres from the intermediate branch to the soleus. Above this level the fibres of the distal and intermediate branches slowly blended and travelled as a system. The last of the bundles devoted solely to fibres from the distal soleus united with a bundle containing plantar and intermediate soleus fibres 8 mm. above the joint.

The general distribution of the distal soleus group before its participation in anastomoses was posterolateral. It entered the extreme lateral margin of the nerve and after a course of 12 mm. was placed just behind the proximal flexor hallucis bundle. 9 mm. higher the tibialis posterior group entered the nerve lateral to it and 48 mm. below the joint the intermediate soleus group entered posterolateral to it. 3 mm. higher the tibialis posterior and flexor hallucis longus groups commenced to migrate medially over the anterior surface thereby leaving the soleus system at the extreme lateral margin of the flattened nerve. The popliteus group entered lateral to it 30 mm. below the joint and within 3 mm. the distal soleus fibres occupied the posterolateral quadrant owing to a change in shape of the nerve from flattened to oval. They retained this localization until a level 11 mm. below the joint when the fibres commenced to mix with those from the intermediate branch.

Intermediate branch.—The fibres comprising this branch joined the lateral margin of the nerve, just behind the distal branch bundle group, 48 mm. below the joint. This position was retained for 37 mm. when one bundle fused with another from the distal branch, and 1 mm. higher a second received plantar fibres. 1 mm. below the joint the last remaining intermediate soleus bundle fused with a bundle composed of the proximal flexor hallucis fibres. Above this level further mixing occurred with plantar and distal soleus fibres.

Ten millimetres above the joint one of the two remaining funiculi composed of distal and intermediate soleus fibres received tibialis posterior fibres while 2 mm. higher the second was joined by a bundle composed of soleus and plantar fibres. This combined system (soleus, plantar and tibialis posterior fibres) ascended in the posterolateral quadrant being slowly displaced medially by the popliteus-lateral gastrocnemius complex. 17 mm. above the joint the soleus fibres commenced to infiltrate across the posterior surface in a medial direction and towards the anterolateral angle. Within 7 mm. they were present in all quadrants except the anteromedial; posteriorly they were associated with plantar fibres only, but anterolaterally they were intermingled with plantar, tibialis posterior and flexor digitorum and hallucis fibres. 33 mm. above the joint they were still contained in all but the anteromedial segment of the nerve, chiefly in association with plantar fibres posteriorly and plantar and tibialis posterior fibres centrally, while one or two anterolateral bundles also contained flexor digitorum and hallucis fibres. At this level the proximal soleus, medial head of gastrocnemius and sural complex, was placed posterolateral to them and the popliteus-lateral head of gastrocnemius complex lateral. 53 mm. above the joint, distal and intermediate soleus fibres were contained in all bundles with the exception of three anteriorly (plantars and flexor hallucis) and a group posteriorly devoted to the proximal soleus, gastrocnemii, popliteus and sural fibres.

Proximal Soleus and Medial Head of Gastrocnemius

The single bundle composed of fibres to these two muscles joined the posterolateral aspect of the nerve 31 mm. above the joint and, within 7 mm., it was placed posteriorly. The bundle group representing these fibres continued in this position for 15 mm. when one of the group fused with a bundle devoted to sural fibres. Anastomoses with neighbouring bundles rapidly occurred and within a millimetre the proximal soleus and medial gastrocnemius fibres were contained in two bundles located posteriorly; in one they were associated with sural fibres and in another with fibres from the popliteus and lateral gastrocnemius. The former complex remained posteriorly with some of the fibres reaching the centre 81 mm. above the joint while the fibres of the latter infiltrated into the posteromedial quadrant. The proximal soleus and medial gastrocnemius fibres were distributed over the posteromedial quadrant 88 mm. above the joint; in this quadrant they were associated with fibres from the popliteus, lateral gastrocnemius, sural, plantar, tibialis posterior and flexor digitorum and hallucis branches. Above this level the soleus system (all branches) slowly infiltrated through the posterior half of the nerve over which they were widely distributed at a level 135 mm. above the joint. They gradually

spread through the remainder of the nerve until at the sciatic notch only a few bundles remained anteromedially which had not received them.

Sural

The sural fibres shared a common stem of origin with those to the proximal soleus and medial gastrocnemius. The bundle joined the posterolateral aspect of the nerve and rapidly moved posteriorly, adopting a position just lateral to the soleus-gastrocnemius complex with which it entered. After a course of 22 mm. this bundle was joined by one of the soleus-gastrocnemius bundles and above this level the behaviour of the sural fibres has been discussed with the complex with which it became associated.

Genicular Branches

Two branches, distal and proximal, were traced from the knee-joint.

Distal.—The fibres entered the medial aspect of the nerve 34 mm. below the joint and continued for 22 mm. when one bundle fused with another containing plantar fibres; the remaining bundle continued for a further 6 mm. when it also fused with a bundle composed of plantar fibres. The genicular fibres were placed medially as far as a level 10 mm. below the joint when they commenced to infiltrate into the posteromedial quadrant. They were not traced beyond a point 35 mm. above the joint at which level they were associated with the plantar fibres contained in the posteromedial quadrant.

Proximal.—The fibres entered the anterior aspect of the nerve, as four bundles, 86 mm. above the joint. After a course of 4 mm. the bundle group separated into two systems.

(i) One group migrated around the lateral surface of the nerve and after a course of 5 mm. fused with bundles containing plantar, flexor hallucis, distal and intermediate soleus, and tibialis posterior fibres; they remained with this complex as far as the sciatic notch where the system was situated laterally.

(ii) The other group remained anteriorly and, after a course of 8 mm., a few fibres passed to a bundle containing flexor hallucis and plantar fibres; the others continued in an anteromedial position. They were still isolated 202 mm. above the joint where, however, they were located posteromedially with the entering hamstring bundles medial to them. The bundles then commenced to anastomose with those composed of fibres from all branches excepting those from the sural, arterial, flexor degitorum and hamstrings. Within 6 mm. this mixing was completed.

At the sciatic notch the proximal genicular fibres were widely distributed posteromedially with some fibres occupying a relatively localized position laterally.

Hamstring Muscles

Adductor magnus.—The branch shared a common stem of origin with that to the semimembranosus. The fibres entered the nerve as a single bundle 188 mm. above the joint and proceeded for 70 mm. One of the three component bundles at this level fused with a bundle composed of fibres from the plantar nerves, the flexor hallucis longus and the proximal genicular; this compound funiculus was joined by another bundle of adductor magnus fibres 4 mm. further proximally and by the remaining adductor fibres a further 5 mm. higher. Thus 79 mm. after entering the nerve all the adductor magnus fibres were contained in one bundle in combination with fibres from the plantar nerves, flexor hallucis longus and the proximal genicular. This system continued proximally for 83 mm. when one of the two component bundles joined a bundle from the semitendinosus and long head of biceps; the other fused with a bundle from the semimembranosus 15 mm. further proximally. Above this level the adductor fibres continued to the notch, undergoing further mixing along their course with the fibre systems with which they had already become associated. Throughout their course, however, they were placed superficially at either the extreme medial tip of the oval-shaped nerve or at the medial end of its anterior surface.

Semimembranosus.—The fibres of this branch entered the nerve 188 mm. above the joint and ascended in an anteromedial position just in front of the adductor magnus complex. After a course of 174 mm. migration had resulted in one of the two component bundles at that level occupying a superficial mid-anterior position while the other had retained an anteromedial position. 3 mm. further proximally the latter bundle anastomosed with a bundle containing fibres from the plantar nerves, flexor hallucis longus, proximal genicular and the adductor magnus. This compound bundle divided after a course of 3 mm. One of the two divisions proceeded to the sciatic notch, splitting *en route* but avoiding further anastomoses; the other fused, 5 mm. below the notch, with a bundle containing fibres from the plantar nerves, flexor hallucis longus and the other hamstring muscles.

The lateral component ascending the mid-anterior surface entered, 11 mm. below the sciatic notch, a bundle containing fibres from all branches of the medial popliteal nerve with the exception of those from the hamstring muscles. After a course of 7 mm. this bundle divided and the two divisions continued to the sciatic notch without further change.

Long head of the biceps and the semitendinosus.—The fibres supplying these two muscles entered the nerve 73 mm. below the sciatic notch and proceeded to the notch along the medial edge of the nerve or the adjacent portion of the anterior surface. Along their course funicular anastomoses

brought them into combination with, in turn, the following groups of fibres: 29 mm. below the sciatic notch, plantar, flexor hallucis and adductor magnus fibres; 5 mm. below the notch, plantar, flexor hallucis, semimembranosus and adductor magnus fibres and, finally, 2 mm. below the notch, with representatives from all the distal branches excepting the sural, arterial and flexor digitorum longus.

General.—The fibres from the hamstring muscles were confined to the extreme medial margin of the nerve and, superiorly, to the medial half of the anterior surface of the nerve. The fibres from the individual muscles were arranged in various combinations in association with those from branches entering at more distal levels. At the sciatic notch the hamstring fibres were distributed as follows: in eight bundles the fibres were mixed with those from the plantar nerves and flexor hallucis longus; in three they were combined with fibres from all branches with the exception of those from sural, arterial and flexor digitorum longus branches while in two they were intermingled with representatives from all the distal branches.

Constancy of the Medial Popliteal Funicular Pattern

The maximum length of nerve with an absolutely constant funicular pattern was 4 mm.

Localization in the Medial Popliteal Nerve

The localization of branch fibres within the nerve at the sciatic notch, mid-thigh, at the junction of the upper fourth and lower three-fourths, and upper three-fourths and lower fourth of the thigh and at the knee-joint is shown in fig. 2. The intraneural topography at other levels is illustrated in the accompanying table.

The Length of Branches as Independent Bundle Systems Within the Nerves

The relevant information is contained in Tables III and IV. The lengths are those for which the branches could be safely stripped without damaging or interfering with the fibre architecture of the nerve. In this connexion it is to be noted that accurate information can only be provided by microscopic examination of serial sections of the nerve. Some of the significant anastomoses between the members of different bundle groups are so fine that dissection of the nerve alone would fail to reveal them.

Discussion and Summary

In the earlier paper (Sunderland, 1945*a*) the literature was fully reviewed and the significance of the intraneural topography of peripheral nerves was discussed in relation to: (*a*) The nature, extent and peripheral effects of injury to a nerve; (*b*) the rate and order of regeneration following injury; (*c*) the mobilization resection and suture of nerves; (*d*) the regeneration and

prognosis following nerve suture; and (*e*) the selective loss of function following nerve compression. These general features do not require repetition.

(1) The fibres representing individual branches at first pursued an independent course either as a single funiculus or as a compact bundle group in which the funiculi engaged in plexus formations. Over this part of their course the fibres were consequently sharply localized though they did not necessarily maintain the same quadrantic relationship as they ascended since migration sometimes carried them obliquely through or over the surface of the nerve. Examples in point were the migrations of

TABLE III.—LATERAL POPLITEAL NERVE

Branch	*Length in mm. from site of entry to level of participation in first anastomosis with a bundle containing other fibres*	*Number of funiculi*	*Changes in the funicular pattern*	*Maximum length (in mm.) of constant pattern*
Extensor hallucis longus (distal)	57	1	0	57
Extensor hallucis longus (proximal)	30	1	0	30
Extensor digitorum longus (distal)	57	1	0	57
Extensor digitorum longus (intermediate)	17	1 to 5	6	10
Extensor digitorum longus (proximal)	1	1 to 3	3	1
Combined fibres from the distal branch to the extensor hallucis and digitorum longus	15	1	0	15
Tibialis anterior	18	3 to 6	8	8
Distal genicular	28	1 to 3	4	16
Peroneus brevis	82	1	0	82
Cutaneous	82	1	0	82
Combined peroneus brevis and cutaneous	36	1 or 2	1	32
Peroneus longus	71	2 to 5	30	9
Sural communicating	37	1 to 6	8	17
Lateral cutaneous of calf	37	1 or 2	2	16
Sural communicating and lateral cutaneous of calf	144	1 to 6	62	13
Short head of biceps (distal)	187	1 or 2	2	90
Short head of biceps (proximal)	77	1	0	77
Combined fibres to short head of biceps	43	1 or 2	1	36
Proximal genicular	94	1	0	94

the funiculi from the short head of the biceps and those from the sural communicating branch and the lateral cutaneous nerve of the calf. The lengths for which the bundles of individual branches coursed as separate and distinct groups within the nerve are given in the text.

The bundles of adjacent but independent bundle groups subsequently anastomosed, thereby effecting a blending of the constituent fibres of the two originally distinct groups. An extension of this process ultimately resulted in the widespread diffusion and blending of fibres from several sources. The scattering of fibres occurred gradually, however, and only as the intercommunications were repeated at successive levels so that the widespread intermingling and dispersal of fibres was achieved only after the fibres had ascended considerable distances along the nerve. Thus the

TABLE IV.—MEDIAL POPLITEAL NERVE

Branch	*Length in mm. from site of entry to level of participation in first anastomosis with a bundle containing other fibres*	*Number of funiculi*	*Changes in the funicular pattern*	*Maximum length (in mm.) of constant pattern*
Flexor hallucis longus (distal)	54	1	0	54
Flexor hallucis longus (proximal)	131	1 to 4	12	94
Arterial	18	1	0	18
Flexor digitorum longus	93	1	0	93
Flexor digitorum longus and arterial	24	1	0	24
Tibialis posterior	100	1 to 7	53	15
Soleus (distal)	100	1 to 4	20	15
Soleus (intermediate)	37	1 to 5	22	10
Popliteus	43	1 to 5	21	4
Lateral head of gastrocnemius	10	1 to 5	8	4
Combined fibres from popliteus and lateral head of gastrocnemius	35	1 to 4	12	7
Combined fibres from the soleus (proximal branch) and medial head of gastrocnemius	22	1 to 5	12	6
Sural	22	1 to 4	6	10
Distal genicular	22	1 or 2	2	15
Proximal genicular (as a compact group)	4	3	0	4
Adductor magnus	70	1 to 4	9	47
Semimembranosus	177	1 to 8	30	18
Semitendinosus and long head of biceps	44	1 to 4	11	11

localization of any particular branch system was at first discrete, then discrete in combination with other fibres, and finally a predominant one only until fibre-diffusion reached a stage where no localization could be said to obtain.

For these reasons the longer the course of a branch within a nerve, that is the longer the nerve and the more distal the site of entry of the branch, the greater the diffusion and the intermingling of its fibres at proximal levels. When, however, the bundles of a branch, which had a very high origin, commenced to anastomose they did so with bundles which had acquired fibres from most, if not all, of the distal branches. As a result the fibres of such proximal branches were rapidly mixed with those from several sources, but since their intraneural course was too short to permit extensive scattering, they remained relatively discretely localized though in combination. An example in point was provided by the fibres to the hamstring branches of the medial popliteal nerve.

Owing to the length of the leg, all but the hamstring branches ran very long courses within the sciatic nerve so that there was ample time for thorough mixing of the fibres of the distal branches to occur. Thus at proximal levels all bundles of the lateral popliteal division, with the exception of those (1 or 2) devoted to the short head of biceps, contained representatives of all the branches. In the case of the medial popliteal nerve the bundles were arranged in five groups which were constituted as follows:

(i) Fibres from all branches excluding those from the hamstring muscles. Two bundles in this group, however, contained some fibres from the semimembranosus.

(ii) Fibres from all branches excluding the sural, flexor digitorum longus, arterial and hamstrings.

(iii) Fibres from all branches excluding the sural, flexor digitorum longus and arterial.

(iv) Fibres from the plantar nerves, flexor hallucis longus and the hamstrings.

(v) Fibres from the plantar nerves, flexor hallucis, tibialis posterior and the distal and intermediate branches to the soleus.

The genicular branches have been excluded in the above groupings.

(2) When a nerve sustains an injury which disrupts the endoneurial tubes at a level where each or most of the constituent funiculi contains fibres from most or all of the branches it follows that, since the axon sprouts must compete during regeneration for endoneurial tubes below the injury, the number of fibres for each individual branch will have an important bearing on the effectiveness of regeneration. Thus if the fibres of a branch are numerous, there is more chance of some of their regenerat-

ing axons reaching corresponding endoneurial tubes; a contributing factor in this regard is the branching of the axon sprouts which occurs with the commencement of regeneration in the proximal stump. When, on the other hand, the fibres to a branch are few in number, their chances of establishing correct continuity when competing for endoneurial tubes will be correspondingly reduced. Actually it is the percentage distribution of the different branch fibres in the individual funiculi which is the significant factor but it has not been possible to acquire this information. Nevertheless the proportionate cross-sectional area of the entire nerve occupied by the fibres representing the different branches may be accepted as a measure of the influence of this factor. This information has been provided.

(3) The repeated division, anastomosis, and migration of funiculi resulted in a continual modification of the funicular pattern along the entire length of the nerves. In no case, however, did the changes induced in the pattern in this way occur so abruptly that the *entire* pattern was altered within a few millimetres though quite obvious differences were apparent in the patterns at levels this distance apart. The longest section of nerve with a constant pattern was 6 mm. The length over which the pattern remained constant varied from 0·25 mm. to 6 mm. with a strong bias towards the lower ranges. Individual bundles and bundle groups, however, pursued much longer courses.

(4) Details of the behaviour of the bundle groups representing the various branches of the nerve and the intraneural localization of the different branch fibres at various levels are provided in the text and charts. For purposes of comparison the localization reported by other investigators has been summarized in Tables V and VI; the methods employed by them, however, have given inconclusive and unreliable results. Of those who have investigated this nerve, Langley and Hashimoto (1917) and McKinley (1921) were not concerned with the localization of individual branch fibre systems.

The results of electrical stimulation and of partial nerve lesions.—The localized peripheral effects resulting from stimulation of different aspects of a nerve exposed at operation and of partial nerve lesions with a correspondingly restricted localization can be explained, and the conflicting results reported by different investigators accounted for, on the basis of the present findings.

Owing to the dimensions of the sciatic nerve there are areas centrally which are inaccessible to the electrodes and which, in the event of injury, cannot be damaged where the superficial fibres escape. A current sufficiently strong to reach these deep fibres would necessarily excite widespread effects since the superficial fibres would be activated simultaneously; as a result, the effect of the current on the deeper regions would be difficult

to assess. Thus electrical stimulation of a large nerve will only produce a localized effect at the periphery when the fibres are accessible to the electrodes and are, therefore, on or towards the surface of the nerve. The demonstration of fibres in a superficial position by these methods, however, is no proof that they are not present elsewhere in the nerve. Thus, immediately distal to the origin of the hamstring branches, the flexor hallucis and plantar fibres were distributed over all bundles in the medial popliteal division of the sciatic nerve though in what proportion in each bundle it was impossible to ascertain. However, bundles were located superficially around the anteromedial aspect of the nerve which were composed solely of these fibres—elsewhere they were in combination with representatives from either most or all of the remaining branches. It is not difficult to imagine that stimulation of the anteromedial surface of the nerve would result in a response which would be localized to the fields subserved by those fibres—the interpretation that the flexor hallucis and plantar fibres were focalized in that region would not be correct.

It would appear that the levels of stimulation in the studies of Marie, Meige and Gosset (1915) and Kraus and Ingham (1920) and the levels of the partial lesions in the clinical cases investigated by the Dejerines, and Mouzon (1915) were such that the involved groups of fibres were destined for specific branches, and that they were superficial and localized in sufficient numbers to give, in each instance, a relatively local peripheral response or effect. Such results, however, are apt to be misleading in that they fail to provide a true picture of the conditions obtaining at the site of stimulation or injury, or accurate information concerning the processes occurring along the length of the nerve which result in fibre diffusion and changing fibre relationships. Thus Kraus and Ingham were led to conclude, erroneously, from their stimulation experiments that "the course (of motor fasciculi) is a straight one from the point where the nerve has been made up by its contributing segments to the point of offset of the fasciculi as a branch."

McKinley from stimulation experiments on the dog's sciatic nerve, at the level of the ischial tuberosity, concluded that "there is little grouping of the motor fibres into functional groups aside from the separation of the nerve into its two main divisions." This observation is in agreement with our findings.

The results of gross dissection.—Gross dissection is quite inadequate for the purposes of accurately defining the behaviour of the funiculi and their contained fibres since many of the significant funicular anastomoses are so minute that they can only be demonstrated by microscopic examination. These finer interlacings were obviously missed by Compton who concluded that "both tibial and common peroneal trunks contain a plexus

TABLE V.—THE LATERAL POPLITEAL DIVISION OF THE SCIATIC NERVE

Investigator	*Method employed and section of the nerve investigated*	*Anterior tibial division*	*Musculocutaneous division*	*Tibialis anterior*	*Extensors of toes*	*Peronei*	*Cutaneous*
Stoffel ..	Macro - dissection. Popliteal fossa	Lateral	Posteromedial	—	—	—	Posterior and medial
Compton ..	Macro - dissection. Thigh and popliteal fossa	Neck of fibula : anterior tibial, anterosuperior ; musculocutaneous, postero-inferior ; peroneus longus in between Popliteal fossa : anterior tibial, anterior and behind it the musculocutaneous ; peroneus longus, posterolateral ; peroneal cutaneous, posteromedial Mid and upper thigh : anterior tibial, anteromedial ; musculocutaneous, posteromedial and central ; peroneal cutaneous, posterolateral becoming posterior and then medial as it descended ; short head of biceps, anterior becoming lateral as it descended					
Langley and Hashimoto	Macro - dissection. Thigh	Antero-lateral	Variable	Short head of the biceps—anterior becoming lateral as it descended		Variable	—
Goldberg ..	Macro - dissection. Popliteal fossa	Central	Transversely through the centre, postero-lateral, lateral or medial	—	—	—	Peripheral in a medial or posterior position
Dejerines and Mouzon	Clinical and operative findings. Popliteal fossa	—	—	Medial to lateral : tibialis anterior, extensor hallucis longus, extensor digitorum longus, peronei			
Marie, Meige and Gosset	Stimulation. Popliteal fossa	—	—	Anteromedial	Extensor digitorum : anterolateral. Extensor hallucis : lateral	Posterior	—
Kraus and Ingham	Stimulation. (*a*) Gluteal region and upper thigh (*b*) Popliteal fossa	—	—	(*a*) Postero or Antero } lateral (*b*) Lateral	(*a*) Posterior (*b*) Lateral	(*a*) Posterior (*b*) Postero-medial	—

TABLE VII.—PERCENTAGE CROSS-SECTIONAL AREA OF THE MEDIAL AND LATERAL POPLITEAL NERVES OCCUPIED BY THEIR FUNICULI AT SIX LEVELS BETWEEN THE SCIATIC NOTCH AND THE NECK OF THE FIBULA

Specimen	*Sciatic notch*	*Junction of the upper fourth and lower three-fourths of the thigh*		*Mid-thigh*		*Junction of the upper three-fourths and lower fourth of the thigh*		*Mid-popliteal*		*Neck of the fibula*	
		M	L	M	L	M	L	M	L	M	L
1	M31, L39	35	33	36	38	35	46	32	43	35	35
2	M15, L16	17	19	17	32	22	20	24	70	18	25
3	M29, L34	42	52	48	50	51	56	50	49	39	28
4	28	34	43	45	43	42	52	43	65	38	37
5	17	26	45	46	43	46	53	36	38	42	36
6	27	29	31	37	37	41	54	33	61	42	32
7	22	19	22	30	47	43	47	34	50	38	40
8	12	18	21	28	30	28	70	21	24	31	18
9	24	22	29	37	33	22	39	24	28	41	37
10	21	24	29	37	36	34	46	36	46	24	40

M = Medial popliteal nerve. L = Lateral popliteal nerve.

TABLE VI.—THE MEDIAL POPLITEAL DIVISION OF THE SCIATIC NERVE

Investigator	*Method employed and section of nerve investigated*	*Hamstrings and adductor magnus*	*Medial head of gastrocnemius*	*Lat. head of gastroc. and soleus*	*Popliteus*	*Tibialis posterior*	*Flexor digitorum longus*	*Flexor hallucis longus*	*Plantar nerves*	*Cutaneous of the calf*
Stoffel	Macro - dissection. Popliteal fossa	Medial	Posterior with some fibres of the soleus placed antero-laterally		—	Anterior	Anteromedial	Lateral	—	Posterior
Compton	Macro - dissection.	Anteromedial								
	(*a*) Upper thigh ;	—	(*a*) —	(*a*) —	(*a*) Antero-lateral	(*a*) Central extending medially and posteromedially				(*a*) Posterolateral
	(*a'*) Mid-thigh ;	—	(*a'*) Transversely through centre	(*a'*) Transversely through centre	(*a'*) Antero-lateral	(*a'*) Anteromedial				(*a'*) Posterior-peripheral
	(*b*) Popliteal fossa	—	(*b*) Medial	(*b*) Lateral	(*b*) Antero-lateral	(*b*) Central				(*b*) Posterior-peripheral
Langley and Hashimoto	Macro-dissection. Thigh	—	Posterior		—	—	—	—	—	—
Goldberg	Macro-dissection. Popliteal fossa	Medial	Peripheral and posterior; in some, located somewhat medially	Antero- or postero-lateral	Anterior—the relation of individual branch systems to one another varied				Variable. Medial and lateral closely associated but their relative positions varied	Peripheral. Varies from posterior to lateral ; postero-medial in one specimen
Dejerines and Mouzon	Clinical and operative findings. Popliteal fossa	—	Medial	—	—	Lateral	Lateral	—	Medial	Sural : medial. Calcaneal : lateral. Lat. plantar : lateral
Dejerines*	Clinical and operative findings. Gluteal region and thigh	—	From medial to lateral : Deep branch of the lateral plantar ; medial plantar ; gastrocnemius and soleus ; tibialis posterior ; flexors of toes ; superficial lateral plantar ; calcaneal branches							Sural : medial
Marie, Meige and Gosset	Stimulation. Popliteal fossa	—	Posterior		—	Anteromedial	Posteromedial		—	—
Kraus and Ingham	Stimulation.									
	(*a*) Gluteal region and upper thigh	(*a*) Postero-medial	(*a*) Posterior		—	(*a*) Anterior	(*a*) Postero-medial	(*a*) Postero-medial		
	(*b*) Popliteal fossa	(*b*) Medial	(*b*) Posterior		—	(*b*) Variable	(*b*) Medial or antero-medial	(*b*) Medial or postero-medial	(*b*) Flexor digitorum brevis : postero-lateral, other fibres medial	—

*Quoted by Benisty—Clinical Forms of Nerve Lesions, p. 191.

involving in each case the central part of the mid-third. Distal to the plexuses the trunks are made up of unbranched bundles. In the proximal third there is little or no plexiform arrangement. The plexuses are overlaid by a layer of longitudinal bundles forming the direct unbranched upward prolongations of the several nerves given off from the trunks below the plexuses. Distal to the plexus the bundles are identical with the nerves emanating from them. . . . The plexus may be supposed to be largely, if not entirely, composed of motor fibres. The purely cutaneous bundles have little or no connexion with the plexus." This, we have seen, is quite incorrect.

Attention has already been directed to the observation that anastomoses involve first the bundles representing fibres of independent branches, then bundle groups representing different branches and finally bundle groups containing fibres from several sources. Plexus formations, as observed macroscopically, would be more obvious just above the site of entry of a large group of branches since at such levels the bundle groups representing the different branches would be anastomosing. Such areas are found just above the knee and proximal to the site of entry of the hamstring branches. This appears to be the basis of reports that along a nerve there are zones of plexus formation which separate stretches over which the bundles or bundle groups are essentially parallel (Dustin, Compton, Langley and Hashimoto). This study has emphasized that the anastomosis and division of funiculi occur repeatedly along the entire length of the nerve so that the maximum length with an absolutely constant pattern was 6 mm. There are, however, regions adjoining the entry of branches where plexus formations are more intense and extensive, the intensity and extent depending on the number and size of the branches; it is these zones which are so obvious when the nerve is teased and examined macroscopically.

(5) *Significant differences between the medial and lateral popliteal nerves.*—In the case of the lateral popliteal nerve the number of bundles in the distal third of the thigh was frequently reduced to one or two. No such reduction was observed in the medial popliteal nerve.

Previous investigations have shown that the funiculi are more compactly arranged in some regions than in others depending on the looseness and amount of the supporting epineurial framework and the amount of fat which it contains (Sunderland, 1945*c*; Sunderland and Bradley, 1948). The cross-sectional area of the funiculi has been measured with a planimeter at six levels between the sciatic notch and the neck of the fibula, and expressed as a percentage of the total cross-sectional area of the nerve trunk at each level. The values given in Table VII were obtained from ten specimens which were selected from the twenty investigated because a high division of the sciatic nerve permitted a separate study of the two

popliteal divisions. These values may be accepted as a measure of the degree of separation of the funiculi. The observations indicate that the relationship between the cross-sectional area of the funiculi and that of the nerve trunk is subject to considerable variation but that, in general, the bundles are more compactly arranged in the lateral popliteal nerve. They also emphasize the large amount of connective tissue packing which is normally present in nerves.

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LEGENDS TO ACCOMPANY TEXT FIGURES

FIG. 1.—The branch pattern of the serially sectioned specimen of the left sciatic nerve. SB and LB = Short and long heads of the biceps femoris; ST and SM = Semitendinosus and semimembranosus; AM = Adductor magnus; G = Genicular; LCC = Lateral cutaneous of the calf; SC = Sural communicating; MG and LG = Medial and lateral heads of the gastrocnemius; S = Sural; Sol = Soleus; P = Popliteus; A. = Arterial; TP = Tibialis posterior; TA = Tibialis anterior; PL and PB = Peroneus longus and brevis; C = Cutaneous; FHL and EHL = Flexor and extensor hallucis longus; FDL and EDL = flexor and extensor digitorum longus; TAT and TMC = Terminal sections of the anterior tibial and musculocutaneous nerves respectively; PT = Posterior tibial nerve (plantar fibres).

FIG. 2.—The number and arrangement of the funiculi and their fibre composition in sections of the medial and lateral popliteal nerves at the levels indicated. The sections are from the left sciatic nerve; the lateral popliteal nerve is on the left. In each section the lateral margin of the nerve is to the left and the posterior aspect is facing upwards or to the top margin of the page. The identification key to the fibre systems is that provided with the charts of each nerve.

LUMBAR PUNCTURE HEADACHE

BY

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AFTER puncture of the spinal theca, whether for diagnostic aspiration of cerebrospinal fluid or for spinal anæsthesia it is not uncommon for a very characteristic headache to develop. Pain may be more or less generalized over the calvarium but is often especially severe at the back of the head and may spread into the neck, shoulders or back. The neck may feel stiff and sustained contraction of the posterior cervical muscles (neck rigidity) is common. But the feature which is quite invariable and which distinguishes this headache from all others is its reaction to change of posture, its very striking exacerbation when the patient sits up, and its relief when the patient lies down. The headache may begin a few hours to a few days after lumbar puncture, and it usually lasts twenty-four hours or more.

McRobert (1918) suggested that this headache was due to the leakage of c.s.f from the hole made by the needle. Leakage of fluid after lumbar puncture was demonstrated by Ingvar in a patient with tuberculous meningitis, by Heldt (1929), Nelson (1930), and Merritt and Fremont-Smith (1938). That this leakage was the cause of headache was first demonstrated by Jacobaeus and Frumerie (1923) who found in two cases that lumbar puncture during the headache showed a spinal pressure of less than 1·5 cm. in one and about atmospheric in the other. The pressure was restored to 10 cm. by injecting 50 c.c. saline in the first and 35 c.c. in the second, headache being relieved in each instance. Nelson recorded 3 further cases with pressures of 13, 15, and 18 cm. at the first lumbar puncture and 2·5, 2·0, and 5·5 cm. at the second lumbar puncture. Kunkle, Ray and Wolff (1943) and I (1939) have each recorded one further case in which lumbar puncture revealed fluid pressure somewhat or greatly reduced, and in which restoration of pressure relieved the headache. A low c.s.f. pressure in patients with lumbar-puncture headache has also been described by Alpers (1925) and Solomon (1924).

I have now examined 11 cases in 7 of which spinal pressure has been measured. These are the subject of this paper.

The spinal fluid pressure.—When a patient with lumbar-puncture head-

ache is again tapped while in the horizontal position, the moment of piercing the spinal theca is appreciated by the characteristic feel of the needle, but no fluid emerges. To demonstrate that the subarachnoid space has actually been entered it is necessary to attach a syringe and withdraw fluid. If a manometer is now connected in the ordinary way no fluid rises in it, and to measure the pressure it is necessary to attach to the needle a saline-filled rubber tube terminating in a small glass tube. In this way it was found that in 6 of the 7 cases of post-puncture headache, cerebrospinal fluid pressure was at or about zero (Table I). In the seventh case it was higher, 80 mm. c.s.f. The table also shows the final effect of injecting warm sterile physiological saline, and fig. 1 shows the relationship between pressure increase and volume injected in 2 cases. It will be noted that in the 6 cases with very low initial pressures the volume of saline needed to restore normal pressure was of the order of 30 to 50 c.c.; in the seventh case rather less. In each of these 7 cases, restoring the pressure in this way abolished headache, if this were present in the horizontal posture, and prevented its occurrence on sitting up. The relief in most cases was temporary, the headache returning in half to two hours, no doubt owing to continued leak through the hole in the theca.

It seems then that the primary cause of headache is diminution in the volume of c.s.f. in the subarachnoid space and that the deficit is of the order of 30 to 50 c.c.

Effect of posture.—It has already been noted that increase of pain on sitting up and relief on lying down are invariable features of lumbar-puncture headache. They have been conspicuous in all eleven cases. If a patient previously lying horizontal in bed quickly sits up, headache starts or begins to get worse in 3 to 15 sec. and reaches its height in 30 to 60 sec. When after two minutes or so the patient lies down the headache begins to lessen almost at once, and finally disappears or ceases to get better in 1 to 2 minutes. This disturbance producing pain is evidently mechanical and is one which develops relatively quickly as the subject assumes the vertical position, declining equally quickly as the horizontal posture is resumed.

Effect of shaking the head.—In 6 or 7 cases so tested pain was momentarily increased by shaking the head quickly from side to side or nodding. The source of pain would seem to be in the tissue intervening between brain and skull, namely in the meninges, for it is here that the chief stress of such a manœuvre would seem to fall.

Jugular compression.—Digital compression of the jugular veins in the neck increased pain in 8 of 10 cases tested; in the remaining 2 pain was unaltered, or the change was too slight to be distinguished from similar pressure on the neck not involving the jugular veins. In 2 cases, the jugu-

lar veins were compressed while the c.s.f. pressure was being measured before saline injection. In one, fluid failed to rise in the manometer tube, in the other the pressure rose from −1 cm. initially to +3 cm. during the compression.

The pulse beat.—Headache throbbed with the pulse in 7 of the 11 cases.

Carotid artery compression.—Compressing the carotid artery low in the neck relieved the headache in 3 cases out of 6, sometimes on the same, sometimes on both sides of the head. The relief was clearly distinguished from a control pressure on the neck not involving the carotid arteries. Release of the compression produced a striking increase of pain with the first pulse beat. In the other 3 cases carotid and control pressures could not be distinguished.

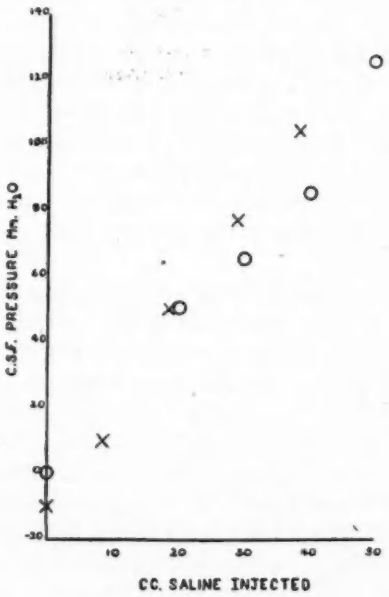

FIG 1.—Shows the relationship between c.s.f. pressure and volume of saline injected in 2 patients with lumbar-puncture headache. Both patients were in the horizontal position and in each case about a minute was allowed for the pressure to level off. Crosses Case 2, 7.11.44, four days after original lumbar puncture. Circles Case 7, 15.11.47, three days after original lumbar puncture.

DISCUSSION.

Kunkle, Ray and Wolff (1943) induced headache in 11 normal subjects by removing about 20 c.c. of cerebrospinal fluid by lumbar puncture in the

erect posture. The headache resembled lumbar puncture headache in being increased by jugular compression and was abolished by restoring the cerebrospinal fluid pressure to normal. This demonstration combined with the low pressures actually found in lumbar-puncture headache and the relief afforded by restoring the pressure provided strong evidence that the headache is due wholly or chiefly to a mechanical disturbance in the cranial cavity. Strictly speaking we should have one further piece of evidence before adopting this explanation of lumbar-puncture headache, namely that equally low pressures do not develop after lumbar puncture in subjects who do not experience headache. This information is at present wanting. Occasionally the cerebrospinal fluid pressure is not so low, 80 mm. in Case 5 of this series and 110 mm. in the case described by Kunkle, Ray and Wolff. In both these cases headache was abolished by raising the c.s.f. pressure by subarachnoid injection of saline. I am not entirely satisfied that in such cases an element of meningeal inflammation can be excluded. Neck rigidity is frequent and it has been shown that in meningitis reduction of c.s.f. pressure below the normal range accentuates headache (Pickering, 1939). Cerebrospinal fluid was not withdrawn for cytological investigation in these cases; Merritt and Fremont-Smith (1938) state that it is always normal in lumbar-puncture headache.

The disturbance would seem to be occasioned by a deficit of some 30 to 50 c.c. c.s.f. in the subarachnoid space, a deficit presumably balanced by an expansion of the venous channels, largely cerebral, of the central nervous system, and by falling in of the less rigidly fixed dura mater of the spinal tube. These changes almost certainly entail a shift in the position of the neuraxis relative to the coverings to which it is attached, and we may suspect that this shift has something to do with initiating headache. Unfortunately little is known as to the volume of c.s.f. in the various compartments of the subarachnoid space, and still less as to the changes in these volumes when the whole system is depleted. The studies of Masserman and Schaller (1933) on the horizontal cadaver indicated that in the skull much of the c.s.f. lies in ventricles and the cisterns surrounding the brain stem. They observed no change after abstracting 10 c.c. at lumbar puncture. Our problem, however, involves larger depletions and particularly their effects in the erect posture. If expansion of cerebral veins is an important factor in compensating for fluid lost there should be a displacement of the brain towards the foramen magnum. We may suspect that any movement of the brain relative to the skull is least over the convexity of the hemispheres and anteriorly and greatest at the base and posteriorly. This conception is in harmony with the observation that in patients with greatly raised intracranial pressure, lumbar puncture may be followed by respiratory failure due to impaction of the medulla

and cerebellum in the foramen magnum. It would be in accord with this train of thought to ascribe lumbar-puncture headache to tension on structures anchoring the brain to its coverings and to expect that the structures in question lie near the base of the brain or posteriorly and are sensitive to deformation. Among such, the tentorium and the coverings of the large arteries of the circle of Willis and its branches at once come to mind.

The observed effects of posture would be in entire harmony with such a conception. For on sitting up the pressure in the lumbar sac rises, and it is probable that this already lax structure expands at the expenses of c.s.f. from the cranial cavity with consequent further caudad displacement of the brain. Such a conception is supported by measurements of lumbar pressure by Nelson. In one case he found at the initial top, pressures of 18 cm. in the horizontal and 53 cm. in the erect postures. When post-puncture headache had developed, pressures were 5·5 cm. in the recumbent, and 28 cm. in the erect posture. The rise of lumbar pressure on sitting up was thus reduced from 35 cm. initially to 22·5 cm. when post-puncture headache had developed. So too with compression of the jugular veins which will increase the volume of blood in the cranial cavity with consequent displacement from it of c.s.f.

Exacerbation of pain with the pulse beat and relief of pain with carotid compression noted in half the cases would also not be surprising if pain were due to tension either on the structures around the circle of Willis or tentorium; for these large vessels pulsate as does the brain, and as I have observed in a brain exposed at operation by Mr. Dickson Wright, carotid obliteration on one side reduces the amplitude of cerebral pulsation though it does not abolish it.

Comparison with the histamine headache.

In an earlier publication (Pickering, 1939) the resemblance between the histamine headache and the lumbar-puncture headache was noted. Both may throb, both are increased by shaking the head, and both are relieved by subarachnoid injection of saline. Jugular compression relieves histamine headache in the normal subject but increases it after spinal drainage,[1] as it does in lumbar-puncture headache. Even carotid compression is not an absolute point of distinction, because, while in severe throbbing headaches after histamine, carotid obliteration produces the most striking relief, in milder headaches particularly in those that do not throb,

[1]When I originally made this observation (Pickering, 1933 - 34) I could not explain it. I now attribute it to the summation of two effects, arterial dilatation and displacement of the brain, each stretching the pain sensitive tissues surrounding the arteries at the base of the brain.

the difference between pressure obliterating the carotid and pressure without effect on the artery cannot be appreciated. This resemblance between lumbar-puncture headache and histamine headache has prompted renewed consideration of the data to see whether any simple explanation would account for the two sets of facts. The evidence against movement of the brain as the chief factor and in favour of stretching of periarterial structures does seem very strong in the case of histamine headache as does the evidence for movement of the brain in lumbar-puncture headache. It seems more likely that the pain in the two instances arises from the same neighbourhood, namely the coverings of the large arteries of the base of the brain, and from the same kind of disturbance, namely tension, though the tension may arise differently in the two instances.

TABLE I.—LUMBAR-PUNCTURE HEADACHE

Shows the c.s.f. pressure measured in the horizontal position in patients with lumbar-puncture headache, and the effects of intrathecal injection of saline.

Case	*Sex and age*	*Diagnosis*	*Neck rigidity*	*Initial press. mm.*	*Vol. saline injected c.c.*	*Final press. mm.*	*Headache relieved*
1	M. 24	Post-traumatic	Yes	0	25	45	Yes
2	F. 59	Bell's palsy	Yes	−10	38	105	Yes
3	M. 53	Hyper-tension	Yes	10	28	140	Yes
4	F. 29	Hysteria	No	0	50	125	Yes
5	M. 45	Post-encephalitic	—	80	20	145	Yes
6	F. 46	Subacute combined degeneration	—	0	30	87	Yes
7	F. 37	Hysteria	Yes	0	43	70	Yes

SUMMARY

(1) In 11 subjects with lumbar-puncture headache, pain often spread to the back of the neck and even into the trunk. Neck rigidity was common.

(2) Cerebrospinal fluid pressure in the horizontal posture was observed to be about atmospheric in 6 cases and within the lower limit of normal in one. In all these 7 cases injection of 30 to 50 c.c. saline, sufficient to restore the pressure to the normal or upper normal range, abolished the headache.

(3) Headache was invariably increased by sitting up and relieved by lying down. It was usually increased by compression of the jugular veins in the neck, and by shaking the head. Not infrequently the headache throbbed with the pulse and in about half the cases was relieved by compression of the carotid artery in the neck.

(4) Lumbar-puncture headache is ascribed to caudad displacement of the base and posterior parts of the brain with tension on the anchoring structures, particularly the tissues around the large arteries at the base.

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OBSERVATIONS ON THE FORAMEN OF MAGENDIE IN A SERIES OF HUMAN BRAINS.

BY

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THE purpose of this paper is to record certain observations on the variations in the size and configuration of the foramen of Magendie in a series of adult human brains. The subject is of interest from the morphological standpoint. Further, these observations may be of significance in the application of encephalography to neurological diagnosis.

It is well known that the 4th ventricle of the human brain communicates with the subarachnoid space through a mid-line foramen of Magendie and lateral foramina of Luschka. The lateral apertures were described by Luschka in 1855 and again by Key and Retzius in 1875. It seems fair to state that the existence of the lateral apertures has never been questioned seriously since they appear to be uniformly present throughout mammals, including man (Rasmussen, 1926, and Strong, Greene and Oliverio, 1926). It will be recalled that lateral recesses of the 4th ventricle wind around the medulla oblongata, passing over the base of the inferior cerebellar peduncle as they do so. These recesses open into the subarachnoid space on the basal aspect of the brain as the foramina of Luschka. Each foramen is situated in the angle between the pons and medulla oblongata and may be identified with ease by the tuft of choroid plexus which all but fills the aperture. The lateral foramina open essentially into the cisterna pontis on the basal aspect of the brain-stem.

The foramen of Magendie, on the other hand, is absent in mammals below the anthropoid apes (Blake, 1900). This fact contributed to a prolonged controversy over the existence of the foramen, numerous workers contending that the foramen in man is merely an artefact produced by tearing the delicate roof of the 4th ventricle during the removal of the brain from the skull or its subsequent manipulation. This controversy was terminated by the advent of human neurosurgery since the foramen may be seen readily at operation. There is no need to revive the argumentative details. They have been reviewed well by Rogers and West (1931) who played a worthy rôle in laying the ghost of artefact. The following general observations will suffice to introduce the material to be recorded in the present paper.

The median aperture of the 4th ventricle was described by François Magendie in 1825. Luschka, Key and Retzius all believed in the existence of the median as well as the lateral apertures of the 4th ventricle. The names of those who took part in the subsequent controversy will not be recorded except to point out that, as early as 1893, Morton described the foramen as seen when the cisterna magna is opened in situ in the cadaver under circumstances which would render tearing of the roof of the 4th ventricle most unlikely. Furthermore, Wilson published photographs of the foramen in 1906. As already mentioned, any excuse for doubting the existence of the foramen in man has ceased to exist with the advent of neurosurgery (Dandy, 1919 and 1921).

It is now well known that the foramen of Magendie is a wide open deficiency in the mid-line of the roof of the 4th ventricle. The 4th ventricle and the cisterna magna of the subarachnoid space are thereby in free communication with each other. In some cases it is difficult to say where the cistern ends and the ventricle begins, so freely do they communicate with each other. In most instances the current textbooks of anatomy illustrate the structure of this region in a satisfactory manner. At the risk of appearing to hold up a specific publication as an example, reference should be made to the accurate drawings of the foramen of Magendie in Mettler's Neuroanatomy (1942). Other very excellent textbooks reproduce a drawing which shows the foramen as a small, punched-out opening, far removed from its true appearance in the actual specimen, except in very rare instances.

There is no need, therefore, to present additional evidence in support of the existence of the foramen of Magendie as a real aperture in the living subject. Numerous excellent descriptions of the foramen in its more usual form are available. It seems appropriate, however, to point out the rather wide variations which occur in the configuration of the foramen and to record the differences in its size in a fairly large series of human brains. These variations may well have a bearing upon the ease with which gas gains access to the ventricular system in the procedure of encephalography.

Material and Methods.

Over a period of several years the region of the roof of the 4th ventricle has been examined carefully in a series of 118 adult human brains which had been removed at autopsy and fixed in formalin. The morphology of the foramen and related structures was noted and measurements of the transverse and mid-sagittal dimensions recorded. Photographs were made in many instances. The examinations were carried out under good illumination and with a binocular loupe. The

arachnoid which forms the posterior wall of the cisterna magna was cut in the mid-line and reflected on the cerebellum. With the greatest care and under direct observation the medulla oblongata was depressed to gain a good view of the region of immediate interest. Since this gentle manipulation was carried out with the roof of the 4th ventricle under direct observation it can be asserted that no tearing of the roof occurred during the course of the examination.

Similar observations were made on several brains immediately after their removal from the skull at autopsy and before immersion in the fixative.

Observations.

The variations which were encountered in the morphology of the foramen of Magendie will be described in groups, as a matter of convenience. It will be appreciated that this grouping of morphological "types" is somewhat artificial and that there is a gradation from one variation to the other.

(*a*) The most frequently encountered configuration of the foramen is as follows (fig. 1). The foramen, far from being a punched-out opening with regular outlines, is a wide open deficiency in the roof of the 4th

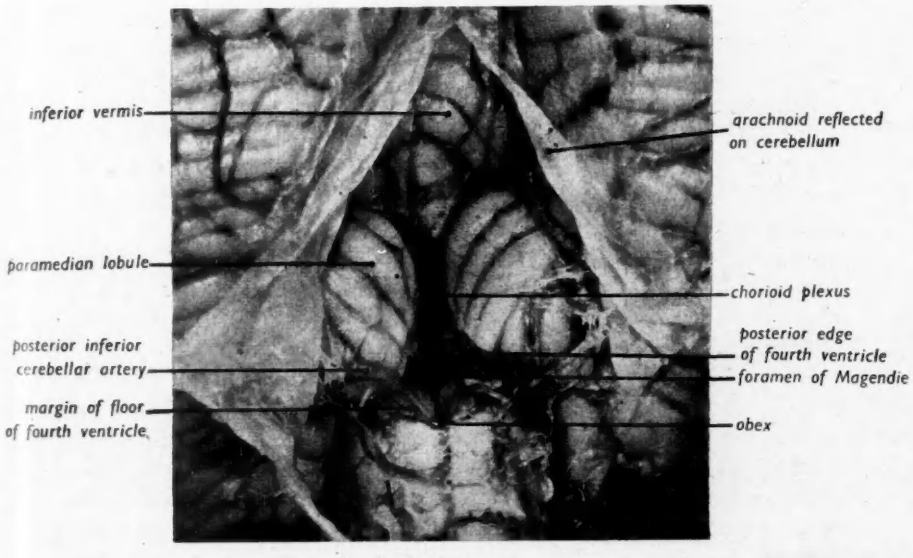


Fig. 1.—A photograph of the roof of the 4th ventricle and adjacent structures to show the morphology of the foramen of Magendie as it is most frequently encountered.

ventricle. The margins of the foramen are formed by the posterior border of the thin pia-ependymal roof and the lateral margins of the floor of the 4th ventricle. The pia-ependymal layer is much abbreviated in most cases. It is attached to the lateral margin of the floor of the 4th ventricle below. The posterior limit of attachment may be anywhere from the region of the obex to a point as much as 8 mm. anterior to the obex. Most commonly the posterior point of attachment is 4 or 5 mm. in front of the obex. The thin pia-ependymal roof is reflected upwards toward the inferior vermis between the paramedian lobules. It may be attached to the pia mater which covers the medial surfaces of the paramedian lobules. On reaching the inferior vermis the roof merges with the pia mater on this cerebellar surface. The posterior border of the pia-ependymal roof may not be as clearly defined as the above description would imply. The posterior portion of the membrane may be fenestrated and merge into the trabeculæ which traverse the cisterna magna from the medulla oblongata to cerebellum. The choroid plexus is suspended from the whole or the more anterior portion of the roof. The plexus is continued posteriorly between the paramedian lobules as progressively more attenuated strands attached to the inferior vermis.

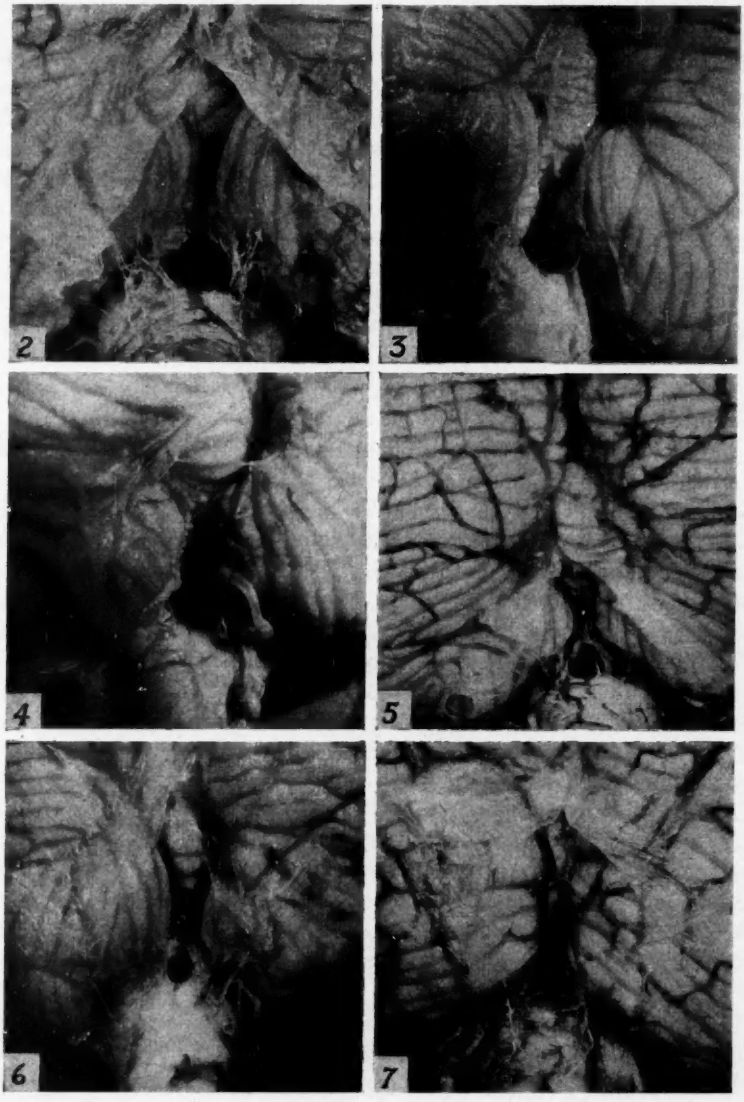
A foramen of Magendie, as described above and illustrated in fig. 1, occurred in 56 per cent of the brains of this series.

(*b*) The most common variations from this standard morphology are due to differences in the extent to which the roof is attached to the inferior surface of the cerebellum. These variations fall into two groups, as follows:

(i) in 23 per cent of the specimens the pia-ependymal membrane does not extend posteriorly along the inferior surface of the cerebellum for any appreciable distance (fig. 5). This portion of the roof of the 4th ventricle is, therefore, relatively flat with the choroid plexus suspended from it in the usual manner. In these instances the outlines of the foramen of Magendie are better defined and the foramen more closely approaches the illustrations which have long appeared in the textbooks of anatomy.

(ii) in 10 per cent of the specimens examined the choroid plexus is prolonged along the inferior surface of the vermis, reaching considerably beyond the posterior limits of the paramedian lobules. Fig. 3 illustrates a tendency toward this posterior prolongation of the plexus. In other cases this feature was much more pronounced, the plexus reaching almost to the posterior limit of the inferior vermis. In these cases the boundary between the 4th ventricle and the cisterna magna becomes rather arbitrary. A part of the plexus appears to secrete cerebrospinal fluid directly into the cisterna magna rather than into the ventricular system.

PLATE XV.



FIGS. 2 to 7.—A group of photographs of the foramen of Magendie have been arranged roughly in order of diminishing size of the foramen from fig. 2 to fig. 7. References have been made to specific features of the photographs in the text.

The variation in the size of the foramina described in sections (*a*) and (*b*) is considerable but in no case could there be said to be other than a free communication between the subarachnoid space and the 4th ventricle. This statement applies whether one thinks of the outflow of fluid from the ventricular system under physiological conditions or the inflow of gas from the cisterna magna to the 4th ventricle in the procedure of encephalography. The average transverse dimension of the foramen at its widest point is approximately 5 mm. In the mid-sagittal line the average dimension from the obex to the nearest point on the inferior vermis is also of the order of 5 mm. Because of the highly irregular outlines of the foramen it is not possible to arrive at an accurate measure of its area. On the whole it may be said to vary rather widely between limits of 5 and 30 sq. mm. with an average size of approximately 16 sq. mm.

In the variants to follow the roof of the 4th ventricle is more intact than in preceding cases.

(*c*) In 7 per cent of brains studied the membranous roof of the 4th ventricle is complete in the mid-line for a variable distance anterior to the obex (fig. 6). In these specimens the dismensions of the foramen varies between 3 by 3 mm. and 3 by 6 mm. The average size of the foramen is less than in the preceding cases, being approximately 7 sq. mm. In two instances a shelf, partly fenestrated and presumably composed of pia-arachnoid, extended from the margins of the inferior portion of the foramen more or less horizontally to the layer of arachnoid which forms the posterior wall of the cisterna magna.

(*d*) The foramen is exceedingly small in 3 per cent of the specimens (fig. 7). The pia-ependymal sheet forms an almost complete roof between the margins of the floor of the 4th ventricle below and the inferior vermis of the cerebellum above, gaining an attachment to the pia mater covering the medial surfaces of the paramedian lobules. The small aperture is of the order of 1 mm. in diameter.

(*e*) Finally, in a single specimen in the series, no mid-line aperture could be found. The membranous roof of the 4th ventricle extended as an uninterrupted sheet from the margins of the ventricular floor below to the cerebellum above.

The foramina of Luschka were examined in all specimens. As far as could be determined they were present consistently and could be identified readily by the tuft of choroid plexus which projects into the foramen from the lateral recess of the 4th ventricle. Neither were there signs of previous inflammatory reaction in the regions of the cisterna magna and roof of the ventricle in any case. It is difficult to be absolutely certain on the latter point from gross inspection. It seems likely, however, that the

morphological configuration described in sections (*d*) and (*e*) represent the normal condition for these particular specimens rather than a result of previous inflammation of the leptomeninges.

Discussion.

There is, then, a considerable variability in the configuration and size of the foramen of Magendie in the adult human brain. This observation is interesting from the point of view of the embryological development of the foramen and in the application of pneumo-encephalographic methods.

Blake (1900) described the development of a blind tubular outpouching from the membranous roof of the embryonal medulla oblongata. He further described the blind end of the outpouching as undergoing dissolution with subsequent development of the hind-brain in man. This view fits in well with the morphology of the foramen of Magendie in the adult brain. In most instances the cephalic wall of the outpouching appears to be caught up with the developing cerebellum, resulting in the posterior extension of the pia-ependyma and the choroid plexus on the inferior surface of the vermis. In some instances the caudal wall of the protrusion persists in the adult as a continuation of the ventricular roof across the mid-line in front of the obex or even as a shelf of tissue, usually fenestrated, which reaches from the margins of the foramen across the cisterna magna to the arachnoid. The extent of the lateral walls which persists into adult life is variable. This results in the considerable differences which exist in the distance between the obex and the most posterior point of attachment of the membranous roof to the lateral margin of the floor of the 4th ventricle. The adult morphology yields no clue regarding the process responsible for the dissolution of the extremity of the dorsal ventricular protrusion during embryological development in man. The development of a large human cerebellum, with resulting mechanical stretching of the thin membrane, may be a factor. That other mechanisms operate seems likely since a large foramen develops even when the protrusion has not been dragged posteriorly by the growing cerebellum (figs. 5 and 6 as compared with figs. 1, 2, 3 and 4). Finally, it appears that in about 4 per cent of cases the mechanism described by Blake fails to occur. In these instances the membranous roof is complete or nearly so. Key and Retzius (cited by Bradley, 1906) found the foramen absent twice in a series of 100 human brains. This corresponds very closely with the incidence of failure of the foramen to develop in the present series.

One can only speculate on the application of these observations to the practice of encephalography. In general, it may be stated that in one

case out of a hundred gas could not enter the 4th ventricle from the cisterna magna so far as the mid-line opening is concerned. In another 3 per cent of cases the foramen is so extremely small that one would expect gas bubbles to pass through the foramen with a great deal of difficulty, if at all. In still another 2 or 3 per cent of brains a shelf, probably representing a persisting portion of the roof protrusion described by Blake, passes horizontally across the cisterna magna caudal to the foramen of Magendie. Even though fenestrated, this thin shelf may impede the passage of gas from the spinal subarachnoid space to the main part of the cisterna magna into which the mid-line foramen opens. Therefore, as far as the foramen of Magendie is concerned, there is a partial or complete obstruction between the subarachnoid space and the 4th ventricle in approximately 6 per cent of cases.

Robertson (1947), in his excellent study of the mechanisms which operate in encephalography, has demonstrated in models that passage of fluid from the 4th ventricle into the subarachnoid space through the foramina of Luschka may assist materially in the filling of the ventricle by air or oxygen entering the ventricle by way of the foramen of Magendie. Whether gas can enter the 4th ventricle through the foramina of Luschka in either the presence or absence of the foramen of Magendie appears to be inconclusive. On strictly morphological grounds, bearing in mind the narrow, tortuous nature of the lateral recesses of the 4th ventricle and the fact that the recesses and the foramina are all but filled with choroid plexus, it appears unlikely that the foramina of Luschka would be of much importance as a direct means of gas entry into the ventricular system. Whether this supposition is entirely true or not must be determined by studies on the dynamics of the gas entry mechanisms in encephalography.

SUMMARY.

The region of the foramen of Magendie has been studied macroscopically in a series of 118 adult human brains removed at autopsy. In 89 per cent of the specimens the foramen is of variable but considerable size, with an average size of 16 sq. mm. In 7 per cent of the specimens the membranous roof of the 4th ventricle is more intact than usual but the foramen is still 7 sq. mm. in size, on the average. In 3 per cent of this series the roof is almost complete, the foramen being represented by a minute aperture a millimeter in diameter or less. Finally, in a single brain a foramen of Magendie is lacking. The possible implications of these observations in encephalography are discussed.

The photographs were prepared by Mr. W. J. T. Austin, Art Service, University of Western Ontario.

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PROJECTION OF THE CENTROMEDIAN NUCLEUS OF THE HUMAN THALAMUS.

BY

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THE centromedian nucleus of primates is agreed by all recent workers in this field to be devoid of projection to any part of the cerebral cortex. A number of investigators have discussed possible projection to subcortical centres. Thus Walker (1938) and Mettler (1945) argue from their evidence (from monkeys) that the globus pallidus is probably its main focus of projection. Walker (1935) had previously described two cases considered to exclude projection to the striatum, whereas Mettler (1947) has recently admitted possible inclusion of the striatum in the projection. Again, C. and O. Vogt (1941) and Freeman and Watts (1947) deduce from their human material that the striatum (putamen plus caudate nucleus) is the chief end-station, each in addition presenting one case estimated to disprove projection to the globus pallidus. Crouch (1940) on the other hand describes wholly intrathalamic projections, and Le Gros Clark (1944) in his most recent communication on the subject appears to favour the conception of the centromedian nucleus as purely a thalmic association centre, though he prefaces his review with the remark: "The centre median nucleus of the thalamus remains a mystery."

The new evidence presented here is derived from human material: from four cases of different kinds of damage to the striatum—vascular (embolic and arteriosclerotic), degenerative (Huntington's chorea), and traumatic (leucotomy)—and from one case of selective degeneration in the globus pallidus (kernicterus). In Cases 1, 2 and 4 comprehensive histological investigation was made of both hemispheres, including serial sections of the basal ganglia and thalamus stained by Nissl's method and a myelin method (Heidenhain) suitable for celloidin-embedded material. In Cases 3 and 5, where sample sections had established the presence of bilaterally symmetrical pathology, only one hemisphere was examined so completely. None but the immediately relevant findings are recorded here.

Case 1.—J. O., male, aged 43 at death, in May 1941, after ligature of the right common carotid artery for gunshot wound, suddenly developed left hemiplegia, presumed to be due to embolism. Thereafter he suffered occasional Jacksonian fits but continued to work until he became depressed and committed suicide by cutting a radial artery in October 1945.

Macroscopically.—The left hemisphere appeared normal in all respects. The convexity of the right hemisphere was extensively shrunken throughout the region of cortical blood supply of the middle cerebral artery (fig. 1). Coronal sections showed the lesion to involve in its depths the head and body of the caudate nucleus and the dorsal aspect of the putamen (fig. 2). The area of supply of the orbital branch of the middle cerebral artery was intact, so that the occlusion had occurred distal to the anterolateral ganglionic arteries.

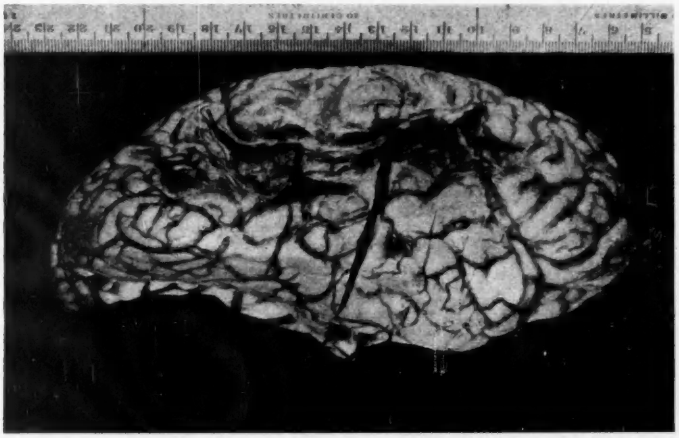


FIG. 1 (*Case* 1).—Ventrolateral aspect of the right hemisphere showing the general extent of the cortical lesion, and the levels of the coronal sections from which fig. 2 is derived.

Microscopically.—The left basal ganglia and thalamus were normal in all respects. Below the ragged surface of the vascular lesion in the right hemisphere there extended a zone of demyelinated and gliosed tissue which gradually merged into the normal brain substance (figs. 3A to D).

The *putamen* was shrunken and had lost about half of its parenchyma, predominantly in the dorsal one-third and along the lateral border. In about half of the remainder the nerve cells were shrunken and the finer network of myelinated fibres had disappeared. Densely proliferated neuroglia replaced the damaged elements.

The *caudate nucleus* displayed loss of nerve cells ranging from a diffuse outfall of about one-quarter in the oral tip to over three-quarters throughout most of the body. In these regions the myelin and glia changes were similar to those in the putamen.

Globus pallidus: At levels of severe involvement of the putamen the lateral division showed demyelination of the large striopallidal bundles and of much of the finer network of fibres. There was only one small area of complete loss of nerve cells, in the dorsal angle over a distance of half a centimetre. The remaining

nerve cells showed considerable excess of lipofuscin. There was a moderate proliferation of both macroglia and oligodendroglia in excess of the normal range. The medial division exhibited no abnormality beyond excess of lipofuscin in a few scattered nerve cells.

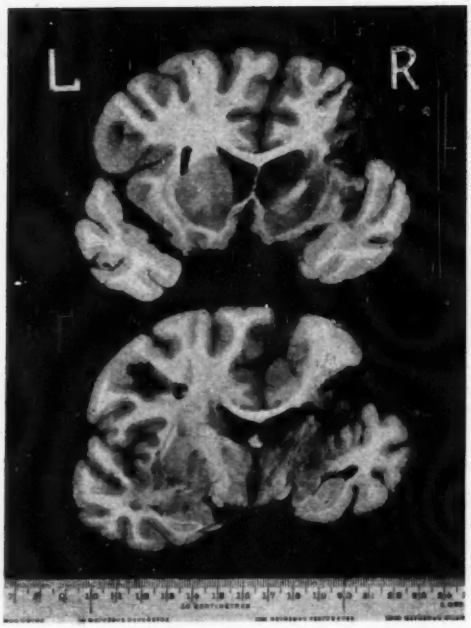


FIG. 2 (*Case* 1).—Coronal sections to display the depth of the old embolic vascular lesion in the right hemisphere.

The *claustrum and capsulæ extrema and externa* were completely degenerated in all except their most ventral parts.

The *internal capsule* was involved in the vascular lesion only in the most dorsal part of the posterior half of the anterior limb.

The *thalamus* was completely free of direct involvement by the vascular lesion, but distorted and severely shrunken. Moderate lipofuscin deposits were present in most cells. Nerve-cell loss and compensatory neuroglial proliferation was in evidence in part of all those nuclei which are known to project to the cortical regions which were destroyed or damaged. Of the remaining nuclei only the centromedian displayed similar changes.

The *centromedian nucleus* was at all coronal levels shrunken to about half the area of its homologue in the left hemisphere. Its myelinated fibres were in consequence about twice as densely packed as in the normal one. It was generally poor in nerve cells and abnormally rich in oligodendroglia and macroglia. The average nerve-cell density per unit field was about 50 per cent of the average in the left nucleus (figs. 4A and B). The remaining nerve cells were moderately loaded with lipofuscin and slightly shrunken. There was no evidence of typical retrograde cell change.

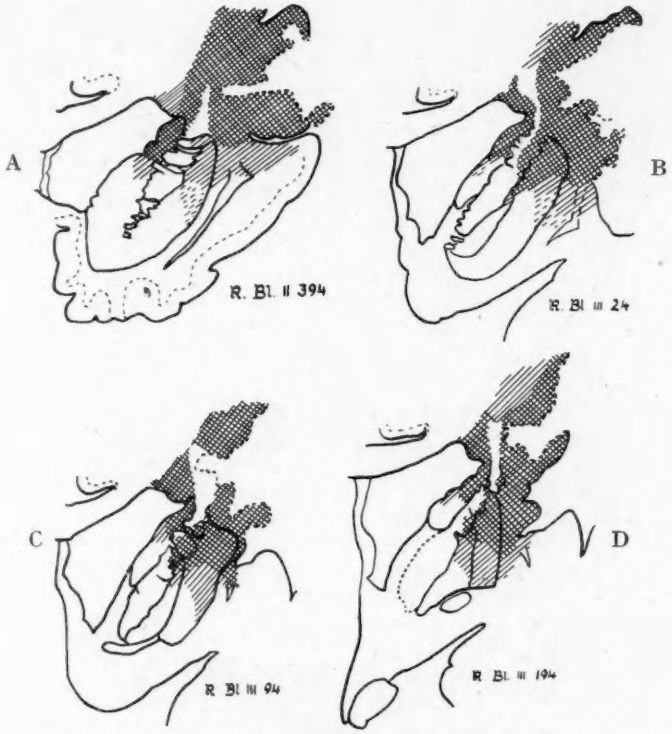


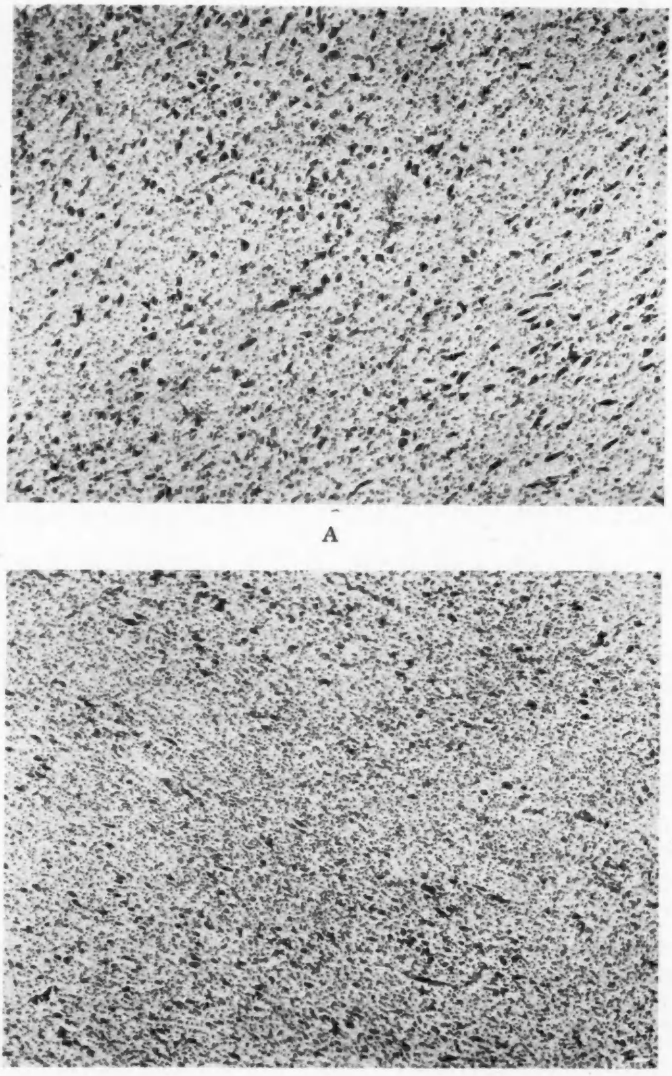
FIG. 3 (*Case* 1).—Diagrams made from stained sections of four representative coronal levels. Cross hatching indicates regions of complete loss of parenchyma, single hatching less complete destruction.

Discussion of Case 1.

The degeneration in the right centromedian nucleus was impressive and was of the same order of magnitude as the destruction of striatal parenchyma. Since cortical connection for the centromedian nucleus has been excluded by previous workers, it would seem beyond reasonable doubt that in this case the destructive striatal lesion, involving damage to afferent axons, was the cause of the degeneration and ultimate disappearance of nerve cells within the centromedian nucleus.

Whether the damage to the claustrum in this case (or Case 2, or Case 4) contributed to the degeneration in the centromedian nucleus is impossible to decide from the evidence available.

That such extensive degeneration in the centromedian nucleus could be accounted for by the very small destructive lesion in the globus pallidus seems highly improbable. The possibility of it being secondary to



A

B

FIG. 4 (*Case* 1).—Homologous regions of A the left, and B the right centromedian nucleus. (Nissl stain. × 70.)

the degeneration of nerve cells within the other thalamic nuclei seems safely excluded by the fact that such atrophy in the centromedian nucleus has never been found in other human cases with severe thalamic

degeneration but without striatal damage (Le Gros Clark and Russell, 1940; Norman, 1945; Sheps, 1945).

This case therefore presents strong evidence of centromedian cell projection to the striatum. It closely resembles Case 1 described by C. and O. Vogt in support of such a connection.

Case 2.—J. G., male, aged 62 at death, had suffered carcinoma of the tongue for seven years with, during the terminal six months, left trigeminal pain for which at first alcohol injections and then medullary tractotomy were carried out. He died on the day after the tractotomy probably due to hæmorrhage from arteriosclerotic vessels. No "extra-pyramidal" symptoms had been noted.

Macroscopically.—The arteries of the base, and especially the anterolateral ganglionic arteries, were tortuous and atheromatous. There was a cherry-sized recent hæmorrhage in the substance of the right cerebellum, but no evidence of gross vascular lesion within the cerebrum beyond an old cystic softening on the lateral border of the left putamen. Both putamina displayed *état lacunaire.*

Microscopically.—There were slight differences on the two sides. The *putamen,* in each hemisphere, contained numerous lacunæ around still persisting medium-sized arteriosclerotic vessels, as well as a wealth of smaller perivascular rarefactions (état criblé). The left putamen (fig. 5) was further damaged, along its ventrolateral border, by a large cyst centred in the ventral half of the external capsule and surrounded by a zone containing large numbers of scavenger cells filled with hæmosiderin.

FIG. 5 (*Case* 2).—Myelin-stained coronal section (× 2·5) of the left basal ganglia, showing the lacunæ in the putamen and the old softening in the external capsule and putamen.

The *caudate nucleus*, on each side, was less severely damaged than the putamen, containing no lacunæ but a number of scattered criblures.

In putamen and caudate nucleus there was, besides these grosser lesions, a general reduction in the number of the small nerve cells. The large nerve cells were rendered conspicuous by heavy deposits of lipofuscin.

The *globus pallidus* in its lateral division, on each side, displayed a few scattered criblures. The nerve cells were diffusely reduced in number, most of the remaining ones being heavily laden with lipofuscin. The medial division was, on each side, essentially normal.

The *claustrum*, on either side, contained a considerable number of lacunæ and criblures, the left one being still further damaged by the cystic softening.

The *capsulæ interna and extrema*, on both sides, were not grossly affected.

The *thalamus*, on both sides, was free of gross arteriosclerotic lesions. The nerve cells were almost everywhere moderately laden with lipofuscin, but there was nowhere any appreciable loss of cells except in the centromedian nucleus.

The *right centromedian nucleus* under low magnification appeared distinctly poor in nerve cells compared with the adjacent nuclei. Higher magnification (fig. 6) revealed irregularly dispersed areas where many nerve cells were in a severe

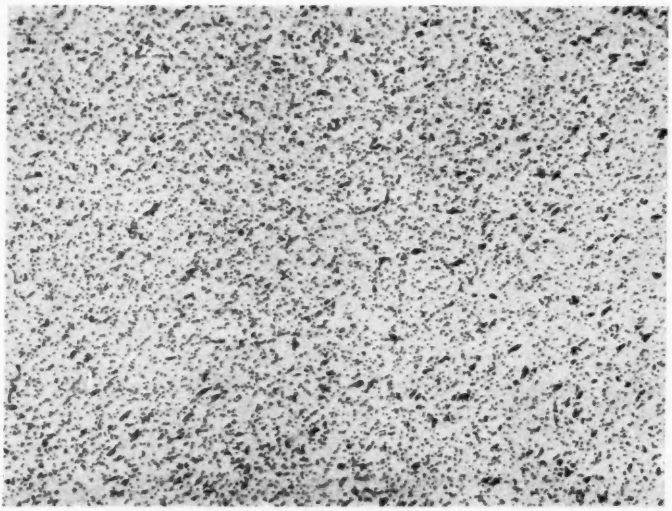


FIG. 6 (*Case* 2).—Right centromedian nucleus: same region as in fig. 4 (Nissl stain. × 70.)

state of pigment atrophy or had completely disappeared. Outside these areas the nerve cells showed varying degrees of shrinkage and less advanced pigment atrophy. There was no evidence of classical retrograde cell change. Both the oligodendroglia and the macroglia appeared proliferated and activated in Nissl-stained sections.

The *left centromedian nucleus* displayed the same general features, but there was in addition degeneration in a large central area throughout which collections of pigment debris around very pyknotic nuclei were the only remains of nerve cells. In this area macroglial proliferation was more intense than in the rest of the nucleus.

Discussion of Case 2.

The destruction of tissue due to arteriosclerosis was much greater in the putamen than anywhere else (except perhaps the claustrum), and was comparable in its intensity, as well as its irregularity of distribution, to the focal degeneration of nerve cells in the centromedian nucleus. Again, the additional large area of cell loss in the left centromedian nucleus seemed to reflect the additional large lesion in the putamen (and claustrum) on this side. These findings therefore strongly suggest direct causal relationship between the centromedian degeneration and the striatal damage.

The causal agent is, as in Case 1, presumably retrograde degeneration, but here the presence of actively extending striatal destruction calls for explanation of the absence of primary irritation in any nerve cells of the centromedian nucleus. It may be that all the nerve cells of the nucleus have passed beyond this stage in retrograde degeneration (*vide* Spielmeyer, 1922), or it may be that centromedian nerve cells, like those of the inferior olive in adult mice (Brodal, 1939), do not pass through such a stage in the course of retrograde degeneration. That the former explanation is more probably correct is suggested by Case 4.

There would seem small reason in this case to attribute the centromedian degeneration to changes in either the other thalamic nuclei or the globus pallidus.

Case 3.—M. B., female, aged 52 at death, had several paternal ancestors who suffered from Huntington's chorea. She developed the disease herself at the age of 44, four years later was admitted to a mental hospital, and continued to deteriorate mentally and physically until her death another four years later from cardiac failure.

Macroscopically.—Coronal sections revealed severe symmetrical shrinkage of the basal ganglia, the caudate nuclei being long and flattened, the putamina reduced to less than half normal bulk and containing a number of small perivascular lacunæ. The globus pallidus on either side was slightly less shrunken.

Microscopically.—The right hemisphere was investigated: In the *putamen* the myelination appeared to be more dense than normal (status fibrosus), although some of the thick bundles did not stain quite as deeply as usual. The small nerve cells were absent throughout. The large nerve cells were unusually densely packed, shrunken and heavily laden with lipofuscin. The macroglia was proliferated and activated, resulting, as seen in Holzer sections, in a fibrous gliosis of moderate degree throughout the nucleus, but of severe degree in the large radial bundles.

The *caudate nucleus* closely resembled the putamen in its myelin, nerve cell and glia picture.

The *globus pallidus* contained no well-defined large myelinated fibre bundles in the lateral parts of its lateral division. Throughout the whole lateral division the nerve cells were considerably reduced in number, the remaining ones being in varying degrees shrunken and laden with lipofuscin; the glial changes in Nissl-stained sections resembled those in the putamen, but were more intense; Holzer staining showed glial fibrosis to be denser than anywhere else in the basal ganglia.

In the medial division, myelinated fibres, nerve cells and glia were practically normal.

The *claustrum* appeared normal apart from the presence of a few scattered nerve cells unusually rich in lipofuscin.

Capsulæ interna, externa and extrema were devoid of focal lesion.

Thalamus: Apart from deposition of a small amount of lipofuscin in most nerve cells and the occurrence of occasional isolated chronic nerve-cell change, all nuclei appeared normal except the centromedian nucleus and the parafascicular nucleus.

The *centromedian nucleus* in low magnification appeared more cellular than normal. Higher magnification revealed this appearance to be due to a moderate increase in both oligodendrocytes and astrocytes, together with a marked shrinkage of the nerve cells (fig. 7) which greatly reduced their normal contrast with the

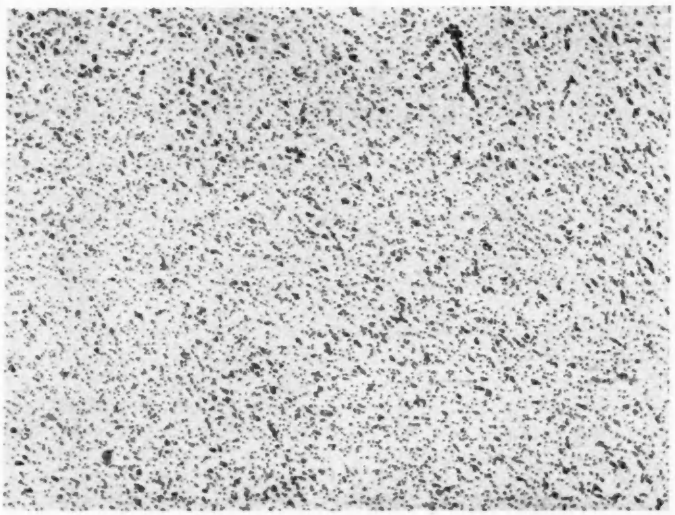


FIG. 7 (*Case* 3).—Right centromedian nucleus, showing marked shrinkage of the nerve cells compared with normal (fig. 4A). (Nissl stain. × 70.)

glial-cell nuclei in Nissl-stained sections (cf. fig. 4*a*). Many of the nerve cells had, in addition, eccentric nuclei and vacuolic cytoplasm-containing lipoids. In places the degeneration had advanced to the stage of disintegration. Classical retrograde cell change was not in evidence.

The *parafascicular nucleus* displayed, especially in its lateral half, similar moderate glial proliferation, but its nerve cells were essentially intact.

Discussion of Case 3.

Such selective degeneration in the centromedian nucleus (or, for that matter, in any nucleus of the thalamus) in Huntington's chorea has never been described in the literature, but the same phenomenon has been found in this laboratory in three other cases of similar clinical and pathological severity. The most conspicuous feature is the generalized

shrinkage and vacuolic degeneration of the nerve cells. To all appearances it belongs to the pigment atrophy type of cell change. Though possibly not differing in kind from the much milder pigment atrophy seen in other regions it is notably more severe in the centromedian nucleus than anywhere else. While there is the possibility of it being due to primary abiotrophy, it would seem more likely that it is secondary to the degeneration in the striatum, for in cases of Huntington's chorea (not yet published) with lesser degrees of degeneration in the striatum the changes in the nerve cells of the centromedian nucleus are correspondingly less severe.

Since, unlike Cases 1 and 2 (and 4), there is here no destructive lesion of the striatum or other acute trauma to axons, the nature of the degeneration in the centromedian nucleus must presumably be of a kind different from ordinary retrograde degeneration. It might be an unusual form of retrograde degeneration due to slow constrictive damage of axons within the gliosing and shrinking putamen. Alternatively, the process might be one of "secondary disuse atrophy" in the sense used by Le Gros Clark and Boggon (1935) in talking of cell shrinkage in nuclei not directly connected with the fibres involved in a lesion.

The significance of the gliosis in the parafascicular nucleus is not clear and requires further investigation.

Case 4.—A. W., female, aged 50 at death, was a paraphrenic who, following bilateral prefrontal leucotomy, developed post-operative restlessness and exhaustion which resulted in a Wernicke syndrome and death four months after the operation.

Macroscopically.—Coronal sections revealed leucotomy lesions as described in detail in "Case 4" of the paper by Meyer, Beck and McLardy (1947). In each hemisphere this traumatic damage involved the dorsum of the head of both caudate nucleus and putamen.

Microscopically.—The serial sections revealed slight differences between the two sides.

In the *left putamen* and *caudate nucleus* the destruction was confined to the oral tip of each nucleus.

In the *right putamen and caudate nucleus* the damage was very slightly smaller and was centred about 1 cm. more caudally, sparing the poles of the nuclei.

The remainder of each putamen and caudate nucleus was normal in cell and myelin picture.

The *globus pallidus* was not directly involved on either side. Apart from increase of lipofuscin in some nerve cells in the dorsal third of the lateral sector, nothing abnormal was discerned.

Claustrum and capsula externa were involved, on both sides, opposite the levels of damage to the putamen.

Capsula extrema was not significantly damaged on either side.

Capsula interna was on each side injured between the damaged caudate nucleus and putamen.

In the *thalamus*, nuclei medialis dorsalis, submedius, ventralis anterior, ventralis lateralis and reticularis on both sides displayed a very considerable loss of nerve cells and compensatory gliosis corresponding to the frontal lobe damage.

The *left centromedian nucleus* was normal in density and appearance of its

nerve cells and glia, except near its dorsomedial angle at about the middle of its antero-posterior length. In that region the eccentricity of the nucleus in some nerve cells was such as to suggest retrograde cell change complicated by the presence of abnormal amounts of pigment content. Other nerve cells in the neighbourhood showed advanced disintegration, others again a striking amount of satellitosis; and the glia, especially the macroglia, was markedly activated.

The *right centromedian nucleus* showed changes similar in every way to those described on the right, but all centred slightly more posteriorly in the nucleus.

In neither nucleus had these changes any resemblance to the typical Wernicke lesions found in the mammillary bodies and paraventricular nuclei of the hypothalamus. The myelin picture was everywhere normal.

Discussion of Case 4.

The circumscribed, although rather ill-defined, degeneration in the centromedian nucleus seems to reflect the limited degree of damage to the striatum, and so to supply supporting evidence, even in such minimal lesions, of centromedian nerve-cell projection to the striatum. Whether this should be taken as evidence of a point-to-point connection is, however, doubtful in view of the poorly defined nature of the lesion in the centromedian nucleus.

The degeneration, as in Cases 1 and 2, is presumably of the retrograde type and here, only four months after the primary lesion, seems to suggest that primary irritation may occur in the early phases of retrograde degeneration of cells in the centromedian nucleus.

This case bears considerable resemblance in its primary lesions to "Case 3" of Freeman and Watts, where the second leucotomy operation (their fig. 8) appears to have destroyed much the same amounts of caudate and putamen as described above, and the centromedian nucleus showed "mild degeneration."

Case 5.—M. C., male, aged 2 years and 1 month at death, had a history of severe jaundice at birth (mother rhesus-negative), and of very retarded development, being unable to speak, sit up or even lift his head by the time of his death from bronchopneumonia.

Macroscopically.—No abnormality was noted beyond shrinkage of the globus pallidus on either side.

Microscopically.—The left hemisphere was investigated. The *globus pallidus* was shrunken and almost completely demyelinated (fig. 8). Less than one-fifth of its nerve cells remained, and all of these were shrunken. The macroglia was activated and proliferated, and glial fibrosis was conspicuous in Holzer-stained sections. In all these respects the medial division was only slightly less severely affected than the lateral division.

The *putamen and caudate nucleus* displayed conspicuous demyelination of both the large radial bundles and the fine network of fibres. The small nerve cells appeared normal, but many of the large nerve cells exhibited a typical picture of primary irritation.

The *claustrum and white matter* were free of abnormality beyond moderate glial fibrosis in the external and extreme capsules.

The *thalamic* nuclei were devoid of significant pathological changes, the centromedian nucleus being no exception.

Other features of note in this case were the complete degeneration of the subthalamic nucleus and considerable degeneration of cells in both the pyramidal layer and the fascia dentata of the hippocampus. In addition, many of the cells of the substantia nigra, which was not yet pigmented, displayed distinct primary irritation.

FIG. 8 (*Case* 5).—Myelin-stained coronal section (× 2·5) of the basal ganglia showing the demyelination of the globus pallidus and the striopallidal fibres in the putamen.

Discussion of Case 5.

The primary irritation in large cells of the striatum and in the substantia nigra requires explanation. Elsewhere in the brain there was no evidence of pellagra. It would seem improbable that these changes had persisted throughout the two years since the jaundice (although the duration of primary irritation is not definitely known). An alternative explanation, though difficult to prove, might be that the changes in these cells were of relatively recent origin due to constrictive injury to the axons of the cells concerned by the gliosis and shrinkage within the pallidum. Whatever the precise mechanism involved, it would appear to have affected all afferent axons to the pallidum. Since no retrograde degeneration whatsoever was found in the centromedian nucleus it seems permissible to conclude that this nucleus does not project to the globus pallidus—the conclusion arrived at by C. and O. Vogt from their case

of status dysmyelinatus, and by Freeman and Watts from their even more convincing case of selective necrosis (due to carbon monoxide poisoning) of the globus pallidus.

Le Gros Clark and Russell (1940) reported similar negative findings in the centromedian nucleus in a case of kernicterus (Case 1 described by FitzGerald, Greenfield and Kounine, 1939) which in its histopathological features closely resembled the present one.

The finding of retrograde cell changes in the substantia nigra is of incidental interest in that it tends to substantiate the existence of nigro-pallidal fibres, a much disputed connection which has recently been discussed by Glees and Wall (1946).

General Comments.

Each of the four cases displaying degeneration in the centromedian nucleus demonstrates improbability that pallidal or intrathalamic changes are responsible for the lesion, and strong probability that striatal damage is the operative factor. Viewed together, therefore, these four cases present very strong evidence that, as C. and O. Vogt and Freeman and Watts maintain, the striatum is the main focus of projection of the cells of the centromedian nucleus, although none of them definitely excludes the possibility that the claustrum shares in the projection.

The fifth case strongly supports the contention of C. and O. Vogt and Freeman and Watts that the centromedian nucleus has no significant connection with the globus pallidus.

Only the general focus of the projection of the nucleus has been considered in this paper. Analysis of its course and more precise terminations is deferred until more material is available for comparison with the detailed point-to-point correlation worked out by C. and O. Vogt.

In conclusion it should be mentioned that throughout this study allowance has been made for the normal variation in cell distribution within the centromedian nucleus, as described by various workers including Le Gros Clark and Boggon and confirmed personally from a study of serial sections of the nucleus in eight normal control cases. The relatively early localized ageing within the centromedian nucleus has also been kept in mind, but none of the present five cases falls within the age-group in which this phenomenon was described by C. and O. Vogt.

Summary.

From a histological study of five human cases with lesions in the basal ganglia evidence is educed that the centromedian nucleus of the thalamus projects to the striatum and not to the globus pallidus.

ACKNOWLEDGMENTS.

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DEMONSTRATION OF A AND C FIBRE COMPONENTS IN THE BABINSKI PLANTAR RESPONSE AND THE PATHOLOGICAL FLEXION REFLEX.

BY

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Introduction.

The afferent fibres in the cutaneous nerves of man include both myelinated and unmyelinated components, comprising a wide range of diameters and conduction velocities. The ratio of myelinated to unmyelinated or C fibres, the major part of which are of dorsal root origin, is about 1:2.

Within the myelinated fibres (mostly A fibres) there is a characteristic segregation into two groups, clearly marked by elevations in the distribution plot of fibres according to diameters. The first elevation is composed of fibres measuring about 16-6 my. The second elevation consists of a narrow band, delta fibres, gathered about a peak at 3-2 my. The findings in man (Ranson, Droegemueller, Davenport, and Fischer, 1935) correspond with those in laboratory animals (*see* e.g. Erlanger and Gasser, 1937; Gasser and Grundfest, 1939).

The Babinski plantar response is generally accepted as part of a general flexion reflex of the lower limb, homologous with the nociceptive flexion reflex (Sherrington) of the lower limb of the spinal and decerebrate animal (Walshe, 1914; Head and Riddoch, 1917), but amongst lower animals the dorsiflexion of the hallux is found only in primates (Fulton and Keller, 1932). In cats Lloyd (1943) showed that the flexion reflex was initiated by two distinct groups of myelinated fibres; the one group corresponding to the first elevation, and the other to the second or delta elevation in the fibre distributive curve of the cutaneous nerve, giving rise to flexion reflexes of characteristically different latencies.

C fibres as the afferent arc in reflex activity have not previously been demonstrated in man, but have been shown in animals giving rise to effects on respiration and blood-pressure (Clark, Hughes, and Gasser, 1935), as well as certain muscular responses (Bishop and Heinbecker, 1935).

In man no attempt seems to have been made to analyse the distribution in the calibre spectrum of the afferent fibres mediating the pathologi-

cal flexion reflex. Clinical experience, however, suggests that the afferents are rather widely distributed, as not only pain, but also such different modalities as warmth, cold, and touch, under favourable conditions, seem to be potent in eliciting the reflex.

The object of the present work is to ascertain which of the above-mentioned three fibre groups in the cutaneous nerve mediate the reflex. This analysis can be facilitated by utilizing the different thresholds of the respective fibre groups for electrical stimulation and conduction velocities (Erlanger and Gasser, 1937), as well as the differences in their sensitivity to the blocking effect of ischæmia and cocaine (Gasser and Erlanger, 1929; Gasser, 1943).

Material and Technique

The *material* on which this investigation is based consists of 15 cases, recorded below:

(1)	Disseminated sclerosis	Spastic paraplegia
(2)	" "	" "
(3)	Progressive spastic paraplegia	" "
(4)	Ginger paralysis	" "
(5)	Infantile hemiplegia	Spastic hemiparesis
(6)	Disseminated sclerosis	Spastic paraplegia
(7)	" "	" "
(8)	" "	" "
(9)	" "	" "
(10)	Myelopathy after spinal anæsthesia	Lost "tendon" reflexes
(11)	Friedreich's ataxia	" "
(12)	Compression of the spinal cord	Spastic paraplegia
(13)	" " "	Paraplegia-in-flexion
(14)	" " "	" " "
(15)	Disseminated sclerosis	" " "

Technique.—The muscles registered were the anterior tibial muscle, a dorsiflexor of the foot, and the long extensor of the great toe, the chief muscle for the dorsal flexion of that toe. The action potentials were led off with pad electrodes from the surface of the skin and, in some cases, with the concentric needle electrodes of Adrian and Bronk (1929). The needle was inserted through an intracutaneous wheal of procaine, in order to diminish the pain. The pain which arises when the needle is moved by the contraction of the muscle is not eliminated. The pain may sometimes be sufficiently intense to elicit or maintain a reflex. In order to eliminate this source of error, needle electrodes, as far as possible, were avoided. They were used only in examination of the long extensor of the great toe, and in some special tests on the anterior tibial muscle. The impulses were recorded with a condenser-coupled amplifier connected to the one beam of a cathode-ray oscillograph. The other beam of the oscillograph was connected to an oscillator which recorded the time.

In natural stimulation, such as a stroke with a finger or needle, or contact with hot metal, the time during which the stimulation had proceeded was recorded on the base-line of the electromyogram, or on the time record. The object causing the stimulation was connected to an amplifier. The changes in voltage on touching of the foot suffice to deflect the rays of the oscillograph if connected to the amplifier.

Skin nerves on the lateral side of the dorsum of the foot were stimulated electrically. Unipolar cathode stimulation was adopted. The cathode was a fine needle, lacquered up to 2 mm. from the point, which was inserted into the nerve. In some tests a pad electrode, 3 mm. in diameter, placed on the surface of the skin, was used instead. In order, in such cases, to avoid stimulation of receptors in the skin simultaneously with the nerve, 1 per cent procaine was injected superficially in the skin beneath the electrode. The anode was a larger electrode fixed on one ankle.

The stimulator gave rectangular current shocks of variable duration (between 0·25 and 8σ) and frequencies up to 300 per second, as well as constant current. In order to avoid distortion of the stimulating current owing to high skin resistance and changes in it due to polarization, the current passed through an amplifier with high internal resistance, 100,000 ohm. The maximum current strength that the stimulator could produce was 17 mA.

Blocking of the cutaneous nerves of the foot with ischæmia in cases where the reflex was registered in the anterior tibial muscle was effected with a pneumatic cuff distended round the instep to 230-300 mm.Hg.

In registration of the long extensor of the great toe, the cuff cannot be placed round the instep, as in that case this muscle, which has its belly far distally, will be paralysed simultaneously with the cutaneous nerves. In such cases the cuff was placed round the upper third part of the leg. The extensors were protected from compression by a plaster-of-Paris bridge under the cuff, raised 2-3 cm. above the skin, and resting on the anterior side of the tibia and fibula. The effect obtained is paralysis of the small muscles of the foot and of the long flexor muscles as well as all grades of skin anæsthesia on the sole of the foot, but without paralysis of the long extensor muscles.

Results

Natural and Electrical Stimulation

If fibres from the entire fibre spectrum are stimulated on the foot, the major part of the discharge in the first elevation of the A group will reach the cord in about 16 msec., and the delta discharge in about 60 msec., reckoned on the basis of a stretch of 1·5 metre and of conduction velocities recorded in experiments on animals (90 and 25 metres per second respectively; *see* e.g. Gasser's chart, 1943). The C fibre discharge, travelling with a conduction velocity of 2-0·6 m.p.s., will reach the cord after about three-quarters of a second to two and a half seconds.

The stimuli, if brief enough, e.g. a condenser discharge if the reflex is mediated by three fibre groups, should give three reflex responses with different latencies: the first two separated by a short interval, and the third coming after some delay. With natural stimulation of the receptors in the skin, e.g. with the prick of a pin or needle, there is small prospect of distinguishing the first two responses. A pin-prick, as one can easily convince oneself, does not cause a short distinct sensation, but pain possibly lasting tens of seconds. In other words, the discharge from the end-organs in the skin outlasts the stimuli. After-discharge in the more rapidly conducting nerve fibres will fill out the interval pending the volley of impulses in the slower A fibres; thus a continuous discharge will reach the spinal cord, and merely a single reflex response will be evoked. Such

stimulation will afford greater prospect of distinguishing between an A fibre and a C fibre response, as they are separated by a much longer interval.

Stroking with a needle.—A light and quick stroking with a knitting needle (fig. 1A) on the sole of the foot will elicit, after 0·4 sec., a reflex

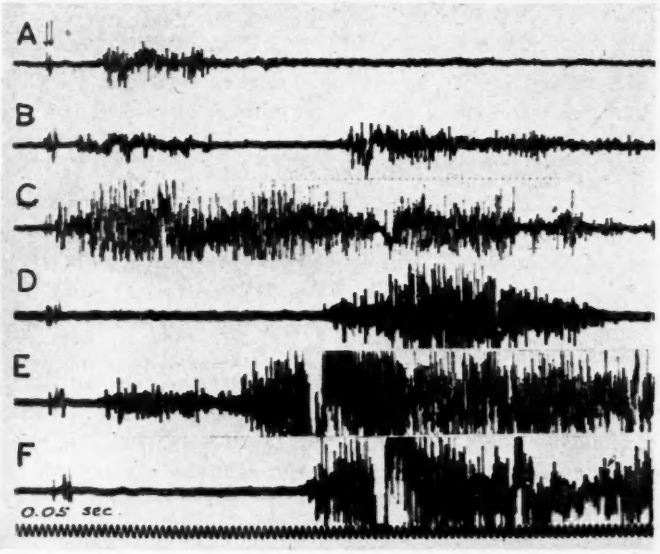

FIG. 1.—Flexion reflex discharge on stroking the sole of the foot with needle. Moment of contact with the needle marked on the base-line of electromyogram (in A between the arrows). This and the following pictures, except 3c, are from the anterior tibial muscle. A. Light stroking; only an early reflex response. B. Moderate stroking; and early and delayed reflex response. C. Strong stroking; one continuous response. D. Moderate contact after 35 min. ischæmia; only a delayed response. E. D.o. Strong stroking; also a weak early response. F. Strong stroking after forty-five minutes ischæmia; only a delayed response.

discharge in the anterior tibial muscle, lasting about ¾ sec. The time that the needle is in contact with the foot is recorded on the base-line of the electromyogram. If the stimulus is somewhat intensified (fig. 1B), the latency will be shortened to 0·2 sec. After another two seconds there will be yet another muscular contraction, observable even with the naked eye, which will last much longer than the first response. Its latency corresponds with that estimated for a C fibre response. If the stimuli is further intensified (fig. 1c), the latency of the first response will be shortened to about 0·1 sec. Merely a single lengthy response can now be observed, due to prolonged after-discharge in the receptors.

Pressure and ischæmia block the A fibres before the C fibres, whilst cocaine blocks the C fibres before the A fibres (Gasser and Erlanger, 1929; Lewis, Pickering, and Rothschild, 1931; Zotterman, 1933; Lewis and Pochin, 1937*a* and *b*; Gasser, 1943). On the assumption that the first response is mediated by the A fibres, and the second reflex response by the C fibres, prolonged ischæmia of the foot will abolish the first response before the second.

A pneumatic cuff is now extended round the instep. After thirty-five minutes' ischæmia (fig. 1D), a stimulus of moderate strength will elicit only the second reflex response. If the stimulus is intensified one can still obtain a weak first reflex response (fig. 1E), insignificant as compared with the very strong and lasting second one. Owing to the ischæmia the threshold for the elicitation of the first response, though previously lower, has now become considerably higher than the threshold for the second response. If the ischæmia is prolonged for ten minutes more, the earlier response will be completely abolished (fig. 1F). At this stage the threshold for the later response is likewise raised. The stimulus must be rather intense in order to elicit a reflex.

This case is representative of a group where the flexion reflex is very easily elicitable: a mere stroke with the finger-tip suffices to elicit it. If the threshold for the elicitation of the reflex is higher, it is difficult to detect any double response. In response to stimulus of moderate strength the reflex will be of short duration, whilst a strong stimulus will cause contractions, possibly of long duration, without a free interval. But, so far as can be judged from observation of three cases, it is possible here also to show a late response, owing to the prolongation of the latency time to one to two seconds after blocking with ischæmia.

The threshold for the first reflex response is usually lower than for the second, when elicited by stroking with a needle. Sometimes, however, the second response can be elicited more easily than the first, especially by a sharp needle on the upper side of the foot where the skin is thin, as shown by Case 11. A well-marked late response, with a latency of 1·5 seconds, is obtained, whereas the first response is missing, or quite slight (fig. 2A). If a somewhat stronger stimulus is applied (fig. 2B), the first reflex response, with a latency time of 0·3 sec., will be more distinct. Ischæmia for forty minutes completely abolishes the first response, although the second response can still be elicited with a maximum strength and duration (fig. 2C).

This case illustrates yet another difference between the two reflexes. The activity of the first one reaches its maximum in some tenths of a second or less. The movement of the limb will therefore be quick. The second response, on the other hand, attains its maximum considerably more slowly, lasts longer, and subsides more gradually. The movement

of the leg, foot, or toe, will therefore be slower and more tonic. This may be due to a difference in the spinal mechanisms of the reflexes, but may also be explained by the differences in the afferent arc, i.e. as an after-

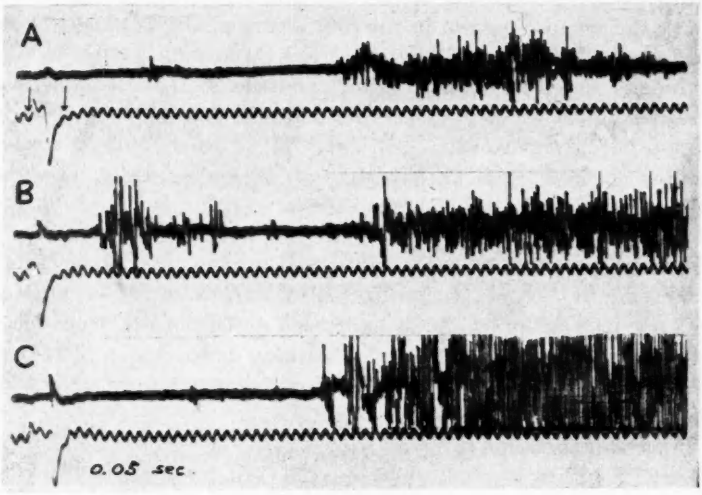

FIG. 2.—Reflex discharge on stroking the upper side of the foot with a sharp needle. Moment of contact of the needle marked on the time record (in A between the arrows). A. Light stroking; a very weak early response and a strong delayed one. B. Moderate stroking; an early response and a much stronger delayed one. C. Strong stroking after forty minutes' ischæmia; only a delayed response.

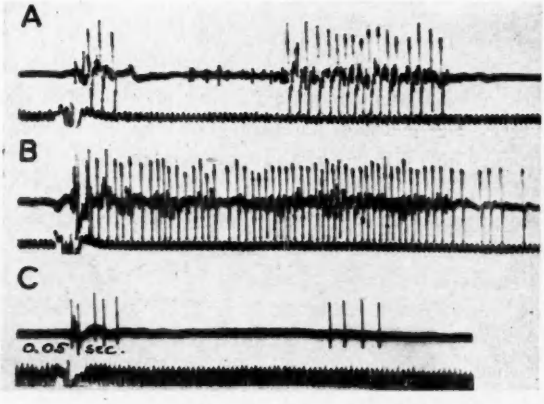

FIG. 3.—Single motor reflex discharge recorded with needle electrodes. A and B from the anterior tibial muscle, C from the long extensor of the great toe. Moment of stimulation with needle stroke marked on the time record. Full description in text.

discharge of longer duration, and a wider spread of the range of conduction velocities in the fibres mediating the second response.

Although there are indications that the reflexes in question in certain respects may have different spinal mechanisms, their final expression as regards the impulse pattern in the contracting muscle is much the same, when analysed with needle electrodes, figs. 3A and B. In this figure the electrode is placed in the tibial anticus muscle, so that the activity of a particular unit is easily observed. In addition, activity from other units can also be discerned on the base-line. These latter units have a lower threshold, being brought into action with weaker muscular contractions, but are more difficult to follow than the large action potential.

The stimulus (fig. 3A) is just over the threshold required to activate the large action potential in the first reflex response. After a latency of about 0·3 sec., the unit repeats three times with a frequency of 10 per second. The same unit reappears after 2 seconds as a late response, repeating with an incipient frequency of 10 per sec., reaching its maximum of 15 per sec. after 0·5 sec., whereupon the frequency again decreases to 10, and suddenly stops.

If the stimuli are increased in strength (fig. 3B), the large action potential, after a latency of about 0·15 sec., will start repeating in two periods, 2 to 3 times, at high frequency. The periods are interrupted by an interval of 0·10 sec., presumably caused by inhibition due to the stretching of an antagonistic muscle (for further details on this point, *see below*). After this, the frequency oscillates between 20 and 10, reaching its minimum 1·5 sec. later. It then again increases, reaching a maximum in about half a second.

The variations in frequency are an index of the development of the excitation process in the motor centre of the reflex. The excitation processes of both reflexes are evidently playing on the same motor neuron, represented by the large action potential shown in the picture. The variations in frequency also reflect the above-mentioned differences between the first and second reflex responses. In the first response the frequency reaches its maximum at once; in the delayed one after about half a second.

In fig. 3C, with the needle in the extensor of the great toe, a unit with the lowest threshold for reflex activation in the picking-up range of the electrode has been selected. It is the large action potential seen in the first response. A smaller one is also recruited, but its threshold is higher. It repeats only once, as against four times for the larger one. The same large spike, as indicated in the fig., has the lowest threshold also in the second reflex response. Consequently, the motor neuron of lowest threshold in both responses is identical, being another example of the similarity in the final motor expression of the two reflexes.

Heat stimulation.—The stimulus that is most certain to elicit the second reflex response is heat. In favourable cases the only response is invariably a delayed flexor reflex on contact with hot metal at a temperature of about 60° C. A short contact at higher temperatures favours the first response relatively to the second one.

In Case 10 (fig. 4A) contact with the shaft of a reflex hammer at 64° C.,

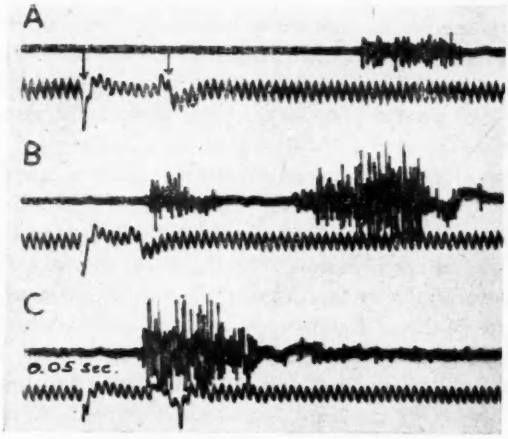

FIG. 4.—Reflex discharge on heat stimulation. Contact recorded on time record (in A between the arrows). A. At 64° C. only a delayed response. B. At 70° C. an early and a stronger delayed response. C. At 70° C. after infiltration of the skin with procaine only the early response.

lasting 0·4–0·5 sec., invariably gave a second response without any trace of a first one. If the temperature is raised to 70° C. (fig. 4B), a first reflex response, simultaneously with the much stronger second response, will be obtained. Infiltration of the skin on the dorsum of the foot with 0·1 per cent procaine within an area of 6 by 6 cm. abolishes the second response (fig. 4C) at a time when a strong first reflex can still be elicited, though its threshold may be somewhat raised.

The latency time for the second reflex response to heat was between 1·3 and 2·3 sec. in ten different cases, being the same as when the reflex had been elicited by the stroke of a needle. The latency is longer for tall persons than for short, and varies in the same person up to half a second according to the intensity of the stimulus. A weak stimulus gives a longer latency than a strong one. The latency evidently varies to some extent also according to the excitability of the spinal cord. The refractory period after a previously elicited flexion reflex, or the inhibition after an extensor reflex, may increase the latency within the above-indicated limits. The

same remark applies to the first reflex response, the latency of which, on stimulation with a needle stroke, varied between 0·08 and 0·5 sec. in fifteen different cases.

To sum up, the view that the early reflex response is mediated in the afferent arc by A fibres, the delayed one by C fibres, is borne out by the reflex times as well as by the order in which they are blocked by ischæmia and procaine.

Now, if it is correct that the double flexion reflex is due to differences in conduction rate, it should be evident that the interval between the two responses must vary with the distance of the point stimulated from the spinal cord. The smaller the distance, the shorter the interval between the reflexes.

To test this hypothesis, a patient with flexion paraplegia (Case 15), where the receptive field of the flexor reflex and the Babinski phenomenon had extended over the whole leg, was selected. In flexion paraplegia the flexion reflex, even after minute stimuli, often breaks into an intense spasm of long duration. In such cases there will be no possibility of grading the stimulus so that a double response is obtained: the result is always one continuous protracted response. In this particular case, however, the double reflex could be elicited with great regularity by stimulation with heat.

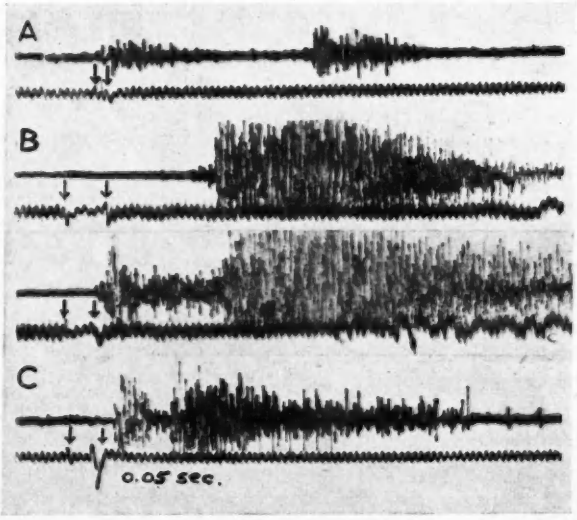

FIG. 5.—Changes in latency of delayed response and the interval between the early and the delayed response, depending on the distance of the point stimulated from the spinal cord. Heat stimulation. A. Stimulus applied to the great toe. B. Just under the knee. C. To the upper part of the thigh.

The stimuli were applied to the great toe (fig. 5A), ·45 cm. proximally thereto just under the knee (fig. 5B) and 32 cm. proximally to the last-mentioned place on the upper part of the thigh (fig. 5C). The fig. strikingly shows how the interval between the early and delayed flexor reflexes dwindles the further proximally the stimulus is applied. The mean for the latency of the second response after stimulation of the toe (6 determinations) was 1·5 sec.; after stimulation below the knee (6 determinations) 1·05 sec.; and on the thigh (6 determinations) 0·6 sec. This corresponds to a conduction rate between toe and knee of 1 m.p.s., between knee and thigh 1·4 m.p.s., values that lie within the range of conduction velocities found for C fibres in animal experiments. The distance from the toe to the corresponding spinal segment was about 1·3 m. With a conduction velocity of 1·2 m.p.s. comes thus 1·1 sec. of the latency (1·5 sec.) on the afferent arc. About 0·4 sec. comes on the central reflex time, a longer value but of the same magnitude as for the early reflex response. The interval of time between the reflexes is thus mainly explained by different conduction velocities in the afferent arc, provided that there has been a strong or moderately strong stimulation.

Electrical stimulation.—The Babinski phenomenon and the flexion reflex are, as is well known, easily elicited by electrical stimulation of the cutaneous nerves with iterative shocks. Single-shock stimulation of short duration (0·25 msec.) is much less effective: 10-30 times higher current strengths are usually required. In many cases 17 mA, the maximal strength of the apparatus employed, has not sufficed. In order to elicit the reflex at lower current strengths, 0·5-5 mA., with single-shock, the duration must be prolonged to 1-5 msec. In such cases the threshold for the excitation of fine nerve fibres will be lower, e.g. Bishop and O'Leary (1939), besides which the nerve will respond with repetitive volleys, e.g. Tureen (1941), which are summed in the centre and facilitate the reflex.

The response to a single shock is a rather asynchronous discharge, the duration of which depends, *inter alia*, on the strength of the stimulation. Some synchronization of the discharge into groups with an interval of 50–250 msec. may, however, occur.

In 6A we see that the discharge in the anterior tibial muscle comes in

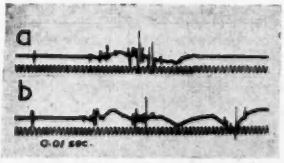

FIG. 6A and B.—Reflex discharge on electrical shock stimulation of a cutaneous nerve. Full description in text.

two groups 0·14 sec. after the stimulation (duration of shock 0·25 msec., strength 5 mA.). When the strength of the stimulus is raised to 7 mA. (fig. 6B), the latency is reduced to 0·125 sec., and 0·25 sec. after the second group, a third group supervenes. Should the strength of the stimulation be increased, or the duration of the shock prolonged, we may have a protracted cyclic discharge with a frequency of 5–10 groups per second, as in clonus.

The interval of 90 msec. between the two groups is brought about by variations in the excitability of the reflex centre, presumably caused by inhibition owing to a proprioceptive inflow from stretched antagonistic muscles of the type described in the classical papers by Walshe (1914, 1923). This phenomenon has been touched on here merely in order to show that we should be cautious in interpreting the two groups of discharge as proceeding from different A nerve fibre flexion reflexes. The duration of the interval in this case would otherwise tally with such an interpretation.

In one case only (No. 9), by extending the duration of the stimulus to σ, a delayed response could be elicited (fig. 7). The threshold for the early reflex response was 1 mA. At 6 mA. (fig. 7A) the response consisted

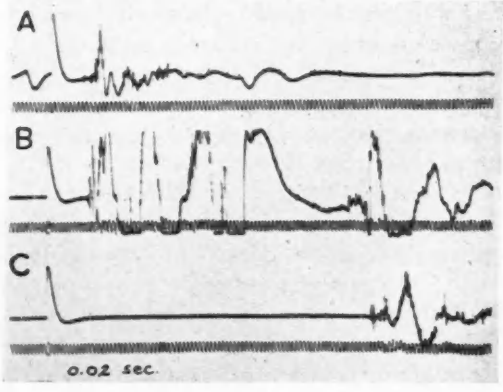

FIG. 7.—Reflex discharge on electrical stimulation of cutaneous nerve with shocks of 3σ duration. Moment of stimulation marked by the shock artefact on the base-line of the electromyogram. A. At 6 mA. a strong early reflex response. B. At 17 mA. a violent early reflex response, and a strong delayed response. (Records badly disturbed owing to the strong muscular contraction which shifts the leading-off cables.) C. Thirty minutes ischæmia; only the delayed response.

of a rather protracted cyclic discharge. At 17 mA. the duration of the very violent reflex increased still further, though not so much as to prevent a second response 2·4 seconds after the stimulation (fig. 7B) being distinctly

observed. Ischæmia for thirty minutes abolishes the first response, leaving the second one intact (fig. 7c).

The stimulating electrode was a fine needle lacquered up to 2 mm. from the point, placed in or close to the cutaneous nerve on the anterior side of the instep. It may perhaps be objected that some other structure than the nerve was then stimulated. However, if the point of the needle was shifted a few millimetres, the threshold for the first response was many times higher, and the delayed one could not be elicited at all. This indicates that the point stimulated really was the nerve.

The Relation of the Early and Delayed Flexion Response to "First" and "Second" Pain

It was noticed at an early date that two distinct flashes of pain could be felt as response to a single stimulus, such as a needle prick or heat (Rosenbach, 1884; Gad and Goldschneider, 1892; Alrutz, 1901; Thunberg, 1902). Zotterman (1933), who determined the latency time for the second pain as Thunberg had done, showed that it corresponded with the computed latency time for impulses travelling in C fibres. His view that the double pain response is due to impulse transmission through fibres of appreciably different conduction rates, the slower ones being identified with C fibres, was strikingly confirmed by Lewis and Pochin (1937*a*).

The correspondence between the latency times found for the second pain as well as its sensitivity to the blocking effect of ischæmia and procaine relatively to the first pain is, broadly speaking, the same as for the delayed flexion response relatively to the earlier one. There can be but little doubt that the impulses giving rise to the second pain and delayed reflex response on the one hand, and to the first pain and early reflex response on the other hand, are, at any rate in part, identical.

To test this view, the patient was told to switch on the ignition as soon as he felt the first or second pain elicited by the same stimulus as induced the flexion reflexes. The times of stimulation, the reflex responses, and the lighting of the lamp are recorded on the same film (fig. 8). In fig. 8A the patient lights the lamp as soon as he feels the second pain, in fig. 8B as soon as he feels the first pain. The patient's reaction time (fig. 8c) was approximately 0·3 sec., being the time required to light the lamp when he observed a deflection on the one cathode ray.

If the reaction time is deducted from the latency time for the second pain, it will correspond, within a few tenths of a second, with the latency time for the delayed reflex response. The same remark applies to the first pain and the early reflex response.

It should further be noted that there is no certain difference in the lag between the early reflex response and the first pain on the one hand, and

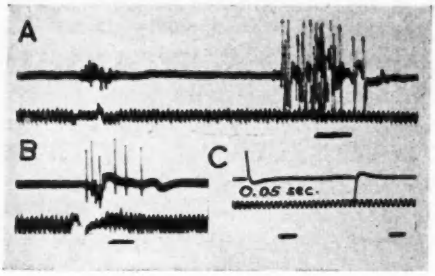

FIG. 8.—The time relation of the early and delayed flexion response to first and second pain. Heat stimulation, marked on time record. The beginning of the black line below the time record in A marks the moment when the patient feels the second pain. In B, the moment of first pain. C. Reaction time of patient. Full description in text.

the delayed reflex and second pain on the other. As the central reflex times of the respective reflexes are of the same magnitude at least on strong stimulation, this must signify that the impulses which give rise to the first and second pain, once they have reached the spinal cord, travel at about the same rate. This indicates, as Lewis and Pochin (1937) have supposed on other grounds, that the delay between the first and second pain arises mainly at the periphery, and argues against the existence in the spinal cord of any special slow-conducting tract connected with the C fibres.

COMMENT

In this paper it is shown that there are at least two flexion reflexes, as well as two extensor plantar responses, with quite separate latencies. There can be little doubt that the explanation of the double reflex response is the transmission of impulses in the afferent arc by fibres with different conduction velocities. The latencies of the reflexes, the reduction of the interval between them, according to the distance from the spinal cord of the point stimulated, their thresholds for electrical stimulation, as well as the order in which the responses are blocked by ischæmia and cocaine, all serve to show that the afferent arc of the rapid reflex may be identified with A nerve fibres, and of the slower one with C fibres.

The wide range of conduction velocities in the afferents is one of the factors in the characteristic slowness and persistence often seen in the execution of the Babinski phenomenon, already stressed by Babinski (1898) in one of the first descriptions of his reflex. In the ordinary clinical test the reflex is first started and maintained with A fibre impulses. It is then continued by C fibre impulses, which do not reach the spinal cord until 1–2 seconds after the A impulses.

Another reason for the persistence of the reflex is that the stimulation gives rise to an after-discharge in the receptors in the skin which maintain the reflex. A third is an after-discharge in the centre. This applies in particular to the spasms most often seen in flexion paraplegia. In this condition, one or more shock stimulations of short duration, causing no after-discharge in the nerve stimulated, may suffice to induce protracted and widespread spasms.

No convincing evidence that the two reflexes have different spinal mechanisms has been adduced. But the second reflex is evidently favoured by the fact that the flexion reflex is very easily elicited, and will then often be more marked than the first. If, however, the threshold for the flexion reflex is high, it may be difficult to elicit the second response, which, if produced at all, will be much weaker than the first. This indicates that the first sign of weakened control from supraspinal levels is the A fibre flexion reflex, the slow response being more easily inhibited and requiring a more complete segmental release from pyramidal control in order to attain its full development.

As regards the A fibre reflex, it has not been found possible to divide it into any components, owing to the difficulty of excluding inhibition by antagonistic reflex systems in single-shock stimulation. Some idea as to which A fibres participate can, however, be obtained by stimulating cutaneous nerves with repetitive shocks.

The fibres that first respond to stimulation are pure tactile (v. Frey, 1896; Heinbecker, Bishop and O'Leary, 1933; Piéron and Segal, 1939). They must have a large calibre, seeing that the rheobase in the ulnar nerve is lower than for the motor nerve fibres (Kugelberg, 1944). Repetitive stimulation of these large fibres apparently does not suffice to elicit the flexor reflex. But, at a current strength of two to three times their threshold value, when the sensation produced is more sharply felt, and is of a rather painful character, they become adequate to evoke the reflex. This will then become stronger according to the intensity of the stimulating current, as tested up to twenty times the threshold for the tactile fibres.

Two to three times the value for tactile threshold is lower than one might expect the threshold for delta-fibre stimulation to be. Heinbecker *et al.* (1933) using induction shocks on excised human and dog cutaneous nerves, found the threshold for the delta potential to be, on the average, five times higher than for the potential with the lowest threshold. Tureen (1941), using condenser shocks on cat nerves, found, in relevant respects, four times the difference in threshold value.

The present author, however, used rectangular shocks of 0·25 msec. duration (frequency up to 200 per sec.), which is not quite comparable with the forms of current used by the above-mentioned investigators. Moreover,

the unreliability of threshold determinations for the tactile fibres in pathological human material must be taken into account. Whether coarser fibres than delta can elicit the reflex is therefore somewhat uncertain. However, to judge from the flexion reflex of experimental animals (Lloyd, 1943), it may be presumed that coarser fibres also participate.

Another interesting point is that the two reflexes can be utilized in order to form an objective test as to what kind of impulses, A or C fibre impulses, are set up by given stimuli. It is, for example, very difficult introspectively to judge whether the nature of the first and second pain is different or not. Lewis (1942) considered that no difference could be shown. Other authors, however, e.g. Zotterman (1933), Bigelow, Harrison, Goodell and Wolff (1945) described the first pain as "pricking," and the second pain as "burning." Bigelow and others, moreover, tested with radiant heat the threshold for pricking and burning pain, and found that the threshold for burning pain was lower. Now the C fibre flexion reflex, if easily elicited, has a lower threshold when evoked by heat stimulation than the first reflex response. This bears out the view that burning pain is conducted by C fibres, and is identical with the second pain sensation.

On the other hand, Wollard, Weddell and Harpman (1939), as well as Bigelow and others, localized the receptive apparatus for the first pain more superficially in the skin than for the second pain. Bigelow and coworkers, moreover, found that scraping the most superficial parts of the skin with a fine needle gave rise to a pricking pain, whilst deeper penetration of the skin produced a burning pain. These observations, based on subjective analysis, correspond well with the fact that the A fibre flexion reflex has a lower threshold for mechanical stimulation of the sole. The C fibre reflex requires a more heavy stroke with a blunt needle, and is favoured by a stroking with a sharp needle in regions where the skin is thin, so that deeper layers are more easily stimulated.

Summary

The afferents in a cutaneous nerve involved in the Babinski plantar response and the pathological flexion reflex have been investigated in 15 human subjects. Two different reflex responses, with quite separate latencies, are noted. The first response is shown to be connected with A nerve fibres, the delayed one with C fibres.

(1) The latency of the early reflex response, depending on the strength of the stimulation, is 0·08–0·5 sec. The discharge generally reaches its maximum more rapidly and subsides more quickly than in the delayed response, so that the action potential patterns in the contracting muscle are otherwise alike. Iterative shock stimulation of the largest tactile fibres in a cutaneous nerve does not elicit the reflex, whereas stimulation at delta fibre strength is effective. Larger fibres than delta may participate.

(2) The latency of the delayed reflex response on stimulation of the foot is 1·3–2·3 seconds. The conduction velocity of the afferents, based on the shortening of the latency by more proximal stimulation, is estimated at about 1 m.p.s. Procaine blocks the delayed response before the early one, whereas the effect of ischæmia is in the reverse order.

(3) The wide range of conduction velocities in the afferent arc is one factor in the characteristic slowness and persistence in the execution of the flexion reflex. Other factors are the after-discharge from receptors, and after-discharge in the reflex centre.

(4) The afferents in the early flexor response are, at least in part, connected with "first pain," and the delayed one with "second pain." The reflexes may be used to show objectively what kind of impulses, A or C fibre impulses, are set up by given stimuli.

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THE MODE OF REPRESENTATION IN THE MOTOR CORTEX.

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ALTHOUGH it has long been established that both motion and inhibition of motion may be produced by stimulation of the cerebral cortex, there is no unanimity in the interpretation of these results. The chief points of disagreement are with respect to the type and extent of functional specialization. The position of different investigators has seldom been explicitly stated but it is possible to reduce the various conflicting interpretations to four mutually incompatible theories. (1) Proponents of extreme specialization hold that each individual muscle has a single point of representation in the cortex. This might be termed the muscle mosaic theory (2). A variant of this view holds that the muscle representation is not punctate but focal; each muscle has a focus or point of maximal excitability which overlaps the representation of other muscles. Muscle representation is denied by other investigators who speak (often rather vaguely) of representation of movement. Simply stated representation of movement means that the response to appropriate electrical stimulation of the cortex does not consist merely of a twitch of a single muscle or part of a muscle but rather has some element of co-ordination such as reciprocal innervation. Movement representation may also be either (3) punctate or (4) focal but even the punctate type implies the possibility of the activation of a number of muscles from any one point.

It was thought possible that an examination of the literature in the light of a careful formulation of these four theories might at least indicate the direction in which a solution might be sought. It was soon found that too often explanations consisted simply of a facile use of words with little regard for the type of nervous organization implied. This is well exemplified by Woolsey's (1947) statement: "It seems unnecessary to insist that movements, not muscles, are represented, or vice versa, since movements are compounded of muscular actions in various combinations."

Theories of representation.—There is, as Walshe (1946) states, "an engaging simplicity" about the punctate theory of muscle representation. This idea considers the motor cortex to consist of a series of mosaics, each

in direct anatomical relation to anterior horn cells, supplying only a single muscle. It would require that if the projection fibres from the motor cortex do not end directly on anterior horn cells then the intervening cells must of necessity be simply relays. The most that such internuncial cells might do would be to make possible the firing of several anterior horn cells by a single corticospinal fibre.

Hines (1947) has recently championed the doctrine of multiple muscle representation. She states that "individual muscles are maximally represented in specific parts of the precentral gyrus—the focus of representation of two muscles was never discovered at exactly the same locus although the fringes of these loci for different muscles overlap to a greater or lesser extent." On this basis stimulation of a point on the motor cortex, under standard conditions, would lead to contraction of a very limited group of muscles with one or two more strongly activated than the others. In structural terms the firing of contiguous cells A, B, and C in the motor cortex would lead to the firing of anterior horn cells a, b, and c whose fibres might go to different muscles. As in the punctate theory this implies that any internuncial cells intervening between the motor cortex and the anterior horn cells are simply relays, all integration is accomplished at the cortical level, indeed, at a premotor level, and the result of cortical stimulation is determined by the point stimulated.

Despite the widespread use of the term movement representation, until recently there had been no attempt to delimit it exactly. Walshe (1947) states that movement "has come to connote, not merely a change in the spatial relations of some part, but also, as underlying such a change, a process embodying some element of neuromuscular integration, of which, perhaps, reciprocal innervation is the most fundamental and constant expression." Punctate movement representation implies a mosaic of points, excitation of each of which produces a different motion. It is difficult to distinguish between such a theory and that of multiple muscle representation. Each would require a fixity of results from threshold cortical stimulation and thus a fixity in connexions between the motor cortex and anterior horn cells. However, multiple muscle representation implies simply that cortical stimulation would produce purposeless activity in a group of muscles, while punctate movement representation suggests the co-ordination in time and space stressed by Hughlings Jackson. In other words, punctate movement representation implies the sequential activity of several muscles in a purposeful manner. It suggests that there is much more to cortical stimulation than the simple activation of Betz cells A, B, and C and the resultant firing of anterior horn cells a, b, and c. For example it implies that there might be a delay between the firing of cells b and c. Just where this delay may occur is difficult to visualize;

yet something of this nature must take place to provide the patterning necessary for a movement rather than the meaningless contraction of several muscles.

It is easiest, and probably best, to define multiple movement representation by a process of elimination. If representation on the motor cortex is not of muscles and if representation is not punctate, there is left only multiple movement representation. This hypothesis requires that the results of cortical stimulation are necessarily determined neither at the cortical level nor by the point stimulated. It further requires that, at threshold strengths of stimulating current, only a portion of the cells at the point stimulated might be fired and that the actual anterior horn cells fired would be the resultant of the impulses carried by the projection fibres from the point stimulated, by the activity in other descending systems and by intrinsic cord activity.

The traditional technique for cortical stimulation.—The charge has frequently been made that results indicating a diffuse representation of either muscles or of movements are artifacts produced by inadequate attention to details of method. Thus suprathreshold stimulation promotes spread of current and multiple responses, primary facilitation may produce reversal of movement, an inadequate interval between periods of stimulation may allow a "cortical march," &c. Such criticism finally led to an almost general adoption of a standardized procedure for cortical stimulation.

This procedure is simple. The strength and duration of a stimulus must be just sufficient to elicit a response and ample time must elapse between successive stimuli so that interaction is impossible. These basic rules (perhaps better called the traditional technique) have been followed in the preparation of the many maps listing the foci from which various responses have been elicited. It is possibly significant that, because there was available a tacitly approved method, discordant results could reasonably be disregarded if this procedure were not exactly followed.

It should be stressed that this standardized procedure will produce a minimum complexity of response regardless of the character of representation. For example, if muscle representation were multiple, widely separated short threshold stimuli would activate a given muscle only when the stimulating electrode was at the focal point for that muscle. Furthermore, the various anæsthetics reduce excitability and to a large extent block integrative activity, and this, again, would lead to a simplification of the response. Certainly, as was stressed by Murphy and Gellhorn (1945), the use of threshold intensities with general anæsthetics may lead to the excitation of only those cortical cells which have the lowest thresholds, and may reveal only a portion of the possibilities of excitation of even this

small number of cells. Similarly the low level of activity in the cord seen under these conditions would greatly limit the available pathways. The standardized conditions would thus favour the elicitation of the type of response predicted by the muscle mosaic theory. However, even in the reports of experiments conducted on this basis there are not infrequent suggestions of variations and discrepancies. Differences between such deviations and the body of fact disclosed by deliberate departures from traditional methods are seemingly of degree only. It is also true that those who have carefully followed the approved methods, impressed primarily by the similarities found, have tended to stress likenesses: the others, impressed by differences, have stressed variation.

Punctate theories not tenable.—It is convenient to consider first the reports available before 1938, for in that year there appeared a text which has had (deservedly or not) much influence upon current neurophysiological concepts. This book was John F. Fulton's "Physiology of the Nervous System." In it there is no explicit discussion of the mode of representation in the motor cortex: there are simply statements of what would be anticipated when the traditional methods were carefully followed. Thus he states (page 402) that "A mosaic of circumscribed foci can readily be demonstrated along the caudal surface of the precentral region from which, on electrical stimulation, isolated contralateral movements, sometimes of single muscles but more often of synergic muscle groups, can readily be obtained; individual responses may be evoked in such muscles as biceps, triceps, flexor longus digitorum and, in man, even a single interosseus may respond." Again (page 410) he states, "Single motor foci of area 4 react to a single stimulus by a short twitch of the affected muscle." There is indirect evidence that some form of punctate theory was favoured for there is no mention of several papers which are opposed to such a theory and no mention of such contradictory evidence in other cited papers. This omission recalls the recent statement of Walshe (1946): "In coming to their conclusions they have left out of account, by a sort of unawareness, all those facts of observation that did not harmonize with the theory; thus giving an example of the insufficiently recognized truth that the relevance of evidence is always dictated by theory; a quality inherent in the observational order of experience."

There is no need to review the many papers which have repeatedly proved that simple isolated activity, often of single muscles, may result from cortical stimulation when the standardized technique is carefully followed. References to practically all such papers may be found in Barker (1899), Sherrington (1909), Lashley (1923), Penfield and Boldrey (1937), Clark and Ward (1937), Scarff (1940), Hines (1943), Walshe (1943), Erickson (1944) and Murphy and Gellhorn (1945*a*). It will be valuable, how-

ever, to consider observations reported in several early papers which are hard to explain under punctate hypotheses.

While adhering fully to the concept of constancy of response to cortical stimulation under standard conditions, Graham Brown and Sherrington (1912) studied some of the factors which tend to produce divergent results. They cut the motor nerves to most of the muscles of the arm so that only the triceps and biceps were active. They then selected a point producing a response in one or the other of the two muscles and stimulated this point repeatedly. At first facilitation and intensification of the response occurred but later a point which at first produced extension might produce flexion. The change could take place either rapidly or slowly and might be shown either by relaxation of one muscle along with contraction of the other or it might be limited in appearance to relaxation alone. Suitable *peripheral* stimulation could reverse a response, for it was found that after such stimulation elbow flexion could be obtained, for example, from a point previously giving elbow extension. Punctate theories would not lead one to anticipate such reversals.

Franz (1915) and Lashley (1923) were both much impressed by the variability they found in the motor cortices of monkeys. Franz (1915) mapped the arm and leg areas in a number of rhesus monkeys and found among the different individuals little correspondence in the location of points from which similar responses could be elicited. Even the two hemispheres of the same brain showed great divergence in localization. These results have been confirmed in man by Penfield and Boldrey (1937), and in the chimpanzee by Leyton and Sherrington (1917).

The differences found are not compatible with punctate representation and are far too extensive to be ascribed solely to anatomical variation or to the limited form of multiple muscle representation recently championed by Chang, Ruch, and Ward (1947).

Lashley (1923) repeatedly stimulated the motor cortex of a small rhesus monkey at intervals of one to fifteen days. At the time of the first stimulation a map was prepared showing topographical features and excitable points determined by bipolar stimulation at threshold intensities. Subsequent stimulations were based on this map. Every effort was made to be certain that the same points were stimulated in the same order and with equivalent strengths of current at each session. In any one test period, the movements obtained from any given point were constant but on other days stimulation of this same point usually induced movements differing from those seen originally. These included not only reversal, such as a change from flexion to extension of the same joint, but also movements affecting other joints or even different body segments. There was no apparent functional relationship between the movements elicited at dif-

ferent sessions so that it is improbable that the results obtained represented portions of co-ordinated muscle patterns. The obvious objection to this report—that the variation was due to errors in placement of electrodes—is not valid. In any one session the response from any single point was unvarying despite the fact that errors of placement were as likely on successive stimulations as on successive sessions. Differences in narcosis were present in successive tests but in any one session a change from very deep to very light anæsthesia produced no change in localization. Since the various points were stimulated in the same order in each experiment, deviation of response (in the sense of Graham Brown and Sherrington, 1912) could not be advanced as the cause for the variation in response. It might be argued that repeated exposure of the cortex (Prados, Strowger and Feindel, 1945) might well produce some tissue reaction but how this could alter responses if connexions between cortex and anterior horn cells were fixed is not understandable. In all essential features this work was confirmed by Boynton and Hines (1933). Penfield and Boldrey (1937) also report results of restimulation of the same individuals. They state, "if the same hemisphere be explored at a later operation after a lapse of time, as we have done in 5 cases, the result may be quite different because areas quite active at the first operation may be mute at the second, and areas which gave no response may later be easily activated." This again represents a confirmation of the essential features of Lashley's report. Such deviations in response are not compatible with any punctate theory.

There was thus available, before the first edition of Fulton's widely quoted text (1938), ample evidence to prove that cortical representation is not punctate and it is quite possible that, as Walshe has implied (1946), such theories were based on an uncritical lack of understanding of the maps of responses elicited that have been published by several investigators. In the past decade, however, there have appeared a number of papers which seem to settle this question of representation.

Movement representation.—In 1938 Ward reported some results of stimulation of the cortex of the unanæsthetized cat by the use of implanted electrodes. Using stimuli of threshold intensity he was able to demonstrate that the pattern of activity in the nervous system might have profound influence on the results of stimulation of one and the same cortical point. He was able to demonstrate flexion as a result of cortical stimulation with the responding limb in the extended position at the onset of the stimulus and extension with the responding limb orginally flexed. It is important to note that the electrodes were fixed and that threshold intensities of stimulating current were used. These results are not compatible with any type of punctate representation nor can they be explained on the basis of the limited multiple muscle representation theory

recently championed by Chang, Ruch, and Ward (1947), and Hines (1947). Recently Clark and Ward (1948) have extended these findings to the monkey. They thought that perhaps the greater spatial separation of motor and sensory areas in the primate would render interpretation of the results less difficult. They were able to demonstrate in the monkey the same phenomena that had previously been demonstrated in the cat (Ward, 1938). With reversal in initial position of a responding limb they were able to demonstrate reversal in response with threshold strengths of stimuli. In view of the recent emphasis on "centripetal individuation"[1] (Hines, 1947) another of their findings is of interest. At threshold stimulation of a fixed electrode the response was a fanning of the toes while with a higher voltage there was only a violent flexion of the knee and hip without movement of the toes. This led them to conclude that strong stimuli may produce a different pattern of response in which the movement elicited by weak stimuli does not occur or is completely masked by other stronger movements. This is not the type of result upon which Boynton and Hines (1933) based their hypothesis. It is difficult to explain the absence of centripetal individuation within the framework of fixed connexions between motor cortex and anterior horn cells. A group of cells excited by low voltage stimulating current would also be excited by one of higher strength. This is especially true if, as Fulton (1943) states, the efferent cells in the motor cortex are directly excited. On the other hand, if the internuncial cells that intervene (Hoff and Hoff, 1934; Lloyd, 1941) between the corticospinal fibres and the anterior horn cells are not simply relays the answer is not difficult. Lorento de Nó (1938*a*, 1938*b*, 1939) has shown how activity in a given fibre might produce diametrically opposite results depending on other concurrent neural activity. He christened such networks "optional transmission circuits." Such a system is very possible in the motor pathway. For example, if cells A, B and C were fired as a result of cortical stimuli, the impulses from these cells would pass to internuncial cells, and we may assume that some of these internuncial cells would be connected to anterior horn cells firing extensor muscles and some to anterior horn cells innervating flexor muscles. With the limb flexed afferent impulses from the stretched extensors might render the anterior horn cells, the fibres of which went to these same extensor muscles, more accessible to impulses from the internuncial cells. In other words, extension would be produced. Similarly with the limb extended, activity in these same corticospinal fibres

[1]The term "centripetal individuation" has been given to the finding that with decreasing strength of stimulating current the response to cortical stimulation becomes increasingly simple and at threshold may be confined to a single muscle (Boynton and Hines, 1933). As has been shown in this review such results apply only to the anæsthetized animal and then only in a single experiment.

might produce flexion. Actual anatomical evidence for optional transmission circuits is not available, but such an explanation was used by Lorento de Nó to explain his results and a similar explanation makes not unreasonable the reversals seen by Ward (1938) and by Clark and Ward (1948).

There is quite a body of recent work which accords with the experiments with implanted electrodes, but which is extremely hard to fit into a punctate or into a multiple-representation-of-muscles schema.

Chang, Ruch and Ward (1947) recorded kymographically the responses to cortical stimulation of muscles acting over the ankle-joint. Even under the restricted conditions of anæsthesia, threshold stimulation and fixation of the limb which they used, evidenced for multiple representation, was obtained. Since this work came from the laboratory that has often afforded strong support for punctate theories of representation and was conducted by the traditional method, it is especially important as evidence against the theories. Their explanations might have been different if they had not failed to note some of the fundamental findings of Tilney and Pike (1925) who introduced the technique. These latter workers demonstrated that an acute lesion of the cerebellum might markedly alter the response to cortical stimulation which is hardly compatible with the muscle representation espoused by Chang, Ruch, and Ward (1947).

Murphy and Gellhorn (1945*a*) deliberately departed from one of the long accepted rules of cortical stimulation. They consistently used a stimulating current above threshold but below convulsive strength. They report that many different movements may be elicited by stimulation of very limited areas of the motor cortex and that there is considerable overlap of the fields from which particular movements may be obtained. Their figures of the extent of the motor cortex from which, for example, flexion of the wrist can be obtained (in a single preparation) remind one strikingly of the figures of Franz (1915) and of Penfield and Boldrey (1937) which were composite figures of the results from several individuals. Murphy and Gellhorn (1945*a*) anticipated the objection that their results were due to spread of current and rather ingeniously showed such an objection to be invalid. It is difficult to harmonize their findings with any type of punctate representation or with the limited multiple representation of muscles recently championed by Hines (1947).

Bosma and Gellhorn (1946, 1947*a*, 1947*b*) using electromyographic techniques have presented convincing evidence that stimulation of a cortical point, even with low intensities of current, produces activity in more than one muscle. In addition, some of their published records reveal that a distinct change in pattern of response may result from a change in strength of stimulating current. This change in some cases was contrary

to that which would be predicted on the basis of centripetal individuation. Equally important is their finding that tonic activity in a muscle or muscle group may profoundly alter a response to a cortical stimulation that involved these muscles. This finding, the earlier one of L. Thompson and Gellhorn (1944), that stimulation leading to nociceptive afferent impulses may modify cortically induced motor responses and that of Murphy and Gellhorn (1945*b*) that hypothalamic stimulation facilitates cortical stimulation all point to the importance of concurrent nervous activity in determining the motor responses to cortical stimulation.

Multiple representation of muscles: The most recent advocate of multiple representation of muscles in Hines (1944 and 1947). She seems to base her belief upon two lines of evidence. She indicates that the small isolated movements produced by threshold stimulation with the proper type of stimulating current proves muscle representation, while the phenomenon of centripetal individuation (Boynton and Hines, 1933) proves that such representation is multiple. Her contention is that the response to threshold stimulation may be restricted to a single muscle while with stronger stimuli progressively more muscles are thrown into action. In other words the response to a strong stimulus represents the addition of responses to those occurring with weaker stimulation. This line of reasoning cannot be accepted for three reasons. First, in the introduction to this paper, it was shown that regardless of the type of representation in the motor cortex, the response to threshold stimuli in the quiescent anæsthetized animal should be small isolated movements. Secondly, Clark and Ward (1948) found that the principle of centripetal individuation does not always hold, and thirdly, it is impossible to fit a reversal in response into the framework of such a motor cortex. Bosma and Gellhorn (1947*b*) have found reversals in response as a result of differences in pre-existing tonic activity, and have shown records of biphasic responses. Neither of these results would be anticipated in a motor cortex organized under the system of multiple muscle representation favoured by Hines. Furthermore, even Chang, Ruch, and Ward (1947) presented kymographic records showing inhibition of one muscle and augmentation in another as a result of an increase in strength of stimulating current applied to a single cortical point. This is not compatible with the theory of centripetal individuation nor with their further remark that their findings call for "no significant revision of Hines' remarkably prescient extrapolations." There is, of course, no doubt that the phenomenon termed centripetal individuation by Boynton and Hines (1933) may occur but also it may not occur, and if the presence of the phenomenon proves that there is multiple representation of muscles in the motor cortex, then its absence must also prove the opposite.

Concluding remarks.—It has been stated earlier that muscle representation, whether punctate or multiple, implies that connexions between the cortex and anterior horn cells are fixed and that the results of cortical stimulation are determined by the point stimulated. It implies that final patterning is accomplished at the cortical level and that any internuncial cells on the way to the "final common path" are simple relays. We have seen that the limited muscle representation favoured by Hines (1947) and by Chang, Ruch and Ward (1947) is not in accord with experimental data. We have further shown that if centripetal individuation is not a universal principle, then integration is not complete at the cortical level and by our earlier definition this is equivalent to saying that muscles are not represented in the cortex. It might seem that Penfield's recent statement (1948) is discordant. He says that "Movement produced by stimulation of the motor cortex takes place most often in those members of the body which are capable of dextrous and complicated voluntary activity, and yet the movements thus produced are never dextrous nor purposeful. Flexion and extension of the arm can be brought about but no patterned action. It is vocalization that follows stimulation, not speech." However, all that could reasonably be expected or integration at either segmental or middle levels (in the sense of Hughlings Jackson's three levels of integration) would be to render the motor activity not discordant with other neural activity. Long ago Lashley (1924)[1] demonstrated the retention of specific learned motor habits after recovery from extensive ablations of area 4, and Loucks (1936) was unable to condition the motor cortex, so at least part of the necessary patterning must occur at still higher levels in order to make possible the complete co-ordination in time and space characteristic of voluntary motor activity.

One may summarize this situation quite briefly. Any particular functional group of spinal internuncial cells receives many, perhaps a majority, of its corticospinal fibres from one small area of the cortex. In addition, fibres from other portions of the motor cortex affect this same group of cells, but the number from each unit area decreases steadily as the distance from the primary focus increases. Stimulation of the motor cortex at threshold levels would excite only those cells in the proper stage of facilitation. Impulses from the cells fired might pass to several groups of internuncial cells but the particular internuncial cells that would be fired by these impulses would be determined by concurrent activity. This concurrent activity would originate in intraspinal afferents, vestibular fibres,

[1]These results of Lashley's (1924) indicate the probability that cortical efferents other than those from the Betz cell area are concerned in learned motor habits and foreshadowed the later proof of a widespread origin of the pyramidal tract (Lassek, 1942; Peele, 1944 and others).

the recently described (Magoun and Rhines, 1948) facilitatory and inhibitory systems, &c. This concurrent activity in accordance with Lorento de Nó's (1938*a*, 1938*b*, 1939) "optional transmission circuits" would summate with corticospinal activity to produce the reversals in response, &c. that have been reported by different workers. This is equivalent to saying that the response to cortical stimulation is not determined solely by the point stimulated and that final integration is not accomplished at the cortical level. In other words, there is neither an individual nor a multiple representation of muscles in the cerebral cortex.

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RESPONSES ELICITED FROM THE CORTEX OF MONKEYS BY ELECTRICAL STIMULATION THROUGH FIXED ELECTRODES

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WHILE there are numerous reports in the literature concerning the effect of electrical stimulation of the cortex in animals under anæsthesia, few exist where the experimental arrangement is such that no general anæsthetic is necessary. Such experiments have been carried out on the cat (Ward, 1938), while Penfield and Boldrey (1937), Rasmussen and Penfield (1947) and Penfield (1947) have reported the results of cortical stimulation in the unanæsthetized human subject. The present experiments constitute a partial analysis of the physiological effects of stimulation of the cortex in unanæsthetized monkeys so that a direct comparison in the several species is possible.

METHODS

The project of stimulating the cortex of the unanæsthetized monkey was made possible because a method for implantation of electrodes in the skull had been developed (Clark and Ward, 1937; Clark, 1941). This method has proved very satisfactory in a number of experiments (Clark and Ward, 1941). Since it has been adequately described elsewhere (Clark and Ward, 1937) only a brief summary is necessary here. Under nembutal anæsthesia, a short incision was made in the scalp and an opening in the skull made with a trephine. The exposed dura was removed and the stainless steel shell holding the electrode was screwed into the skull. A total of 107 electrodes were implanted on 24 hemispheres of 12 immature animals (*macaca mulatta*) and

[1]The actual experiments were all performed in the Department of Anatomy, Vanderbilt University School of Medicine, Nashville, Tennessee, while the senior author was on a short leave of absence. He (G. C.) wishes to take this opportunity to express his appreciation for the many kindnesses and courtesies extended to him during his stay in Nashville and is especially grateful to Dr. Sam L. Clark for the privilege of working in his laboratory and for the constructive criticism given during the progress of this study. The aid of Dr. K. S. Lashley in preparation of the manuscript is also gratefully acknowledged.

these were stimulated a total of 657 times. In all experiments it was intended that the electrodes should overlie the anterior and posterior portions of the motor cortex and in some that the electrodes should be placed on the "eye-fields" as well. The actual sites were selected with reference to the mid-line and the coronal suture (Krieg, 1945). On the day following implantation of the electrodes, the animal was placed in an examination chair and was stimulated with 60 cycle current. An account of the actual results of each stimulation was immediately made by mechanically recorded dictation. The initial current strength was usually 1·0 volt and stimulation was continued for four seconds, unless a violent response occurred. The character of the response, if present, determined the strength of succeeding stimuli

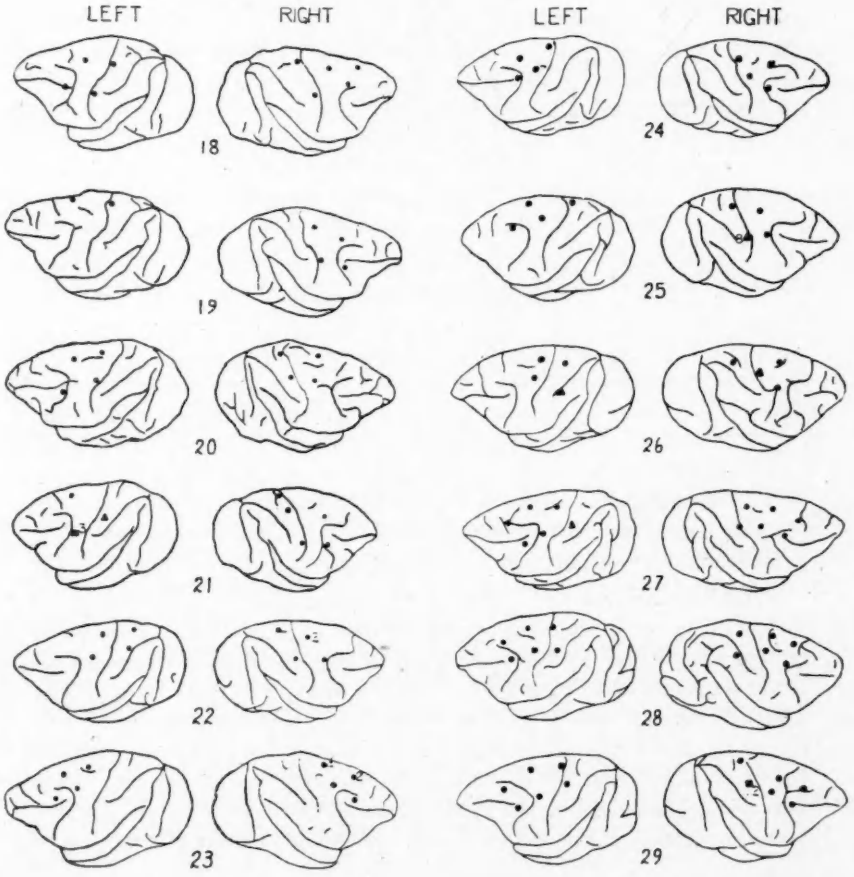

FIG. 1.—Diagrams of hemispheres stimulated with location of electrodes. Animal numbers between and below each pair of hemispheres. Relaxation points indicated by squares, sensory points by triangles. Movements were elicited from all points. Particular electrodes mentioned in text are indicated by a number beside point.

which were often only 0·1 to 0·2 volt higher than those which evoked no response. At the end of each experiment the animal was anæsthetized, killed by bleeding and perfused with 10 per cent formalin. The locations of the electrodes were marked by black bristles inserted into the hardened brain before its removal from the skull.

Drawings of each hemisphere of the twelve animals were made with the aid of a camera lucida and are shown in fig. 1. A composite record of all the points stimulated is given in fig. 2. The outline and fissurization of the latter figure were redrawn from a diagram by McCulloch (1944) to which reference should be made for presumed areal subdivisions.

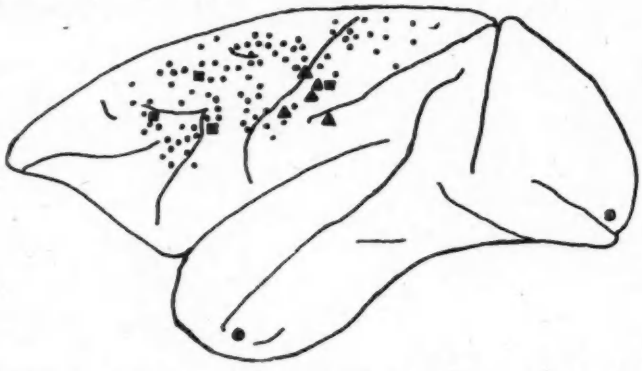

FIG. 2.—Composite diagram indicating location of all points stimulated. Relaxation points indicated by squares, sensory points by triangles. The outline of the figure was redrawn from McCulloch (1944).

RESULTS

The results will be reported under the following categories at this time[1]: (1) The character and thresholds of the responses; (2) evidence for cortically induced sensation; (3) the comparison of the effects of strong and weak stimuli; and (4) cortically induced relaxation.

(1) *Character and thresholds of the response.*—In an unanæsthetized monkey it is not practical to attempt to determine whether a response to cortical stimulation involves contraction of only one muscle, or, indeed, on many occasions, even to determine precisely what muscles are active. Only actual movement will be described. Some of the responses we observed to threshold stimulation involved only a single joint, but more complex responses were common. We found no difference in the character of the responses obtained from the anterior as compared with those from the posterior portion of the motor cortex. Even with threshold strengths of

[1]There are in preparation two further papers based partially upon additional experiments performed on the monkeys reported here. One (Ward, 1948) will deal with the effects of change in initial position of the responding limb on the movement elicited by cortical stimulation and the other (Clark and Ward, 1948) with the results elicited by simultaneous or consecutive stimulation of two electrodes.

stimulating current we were unable to confirm the repeated assertion that stimulation of the anterior portion of the motor cortex produces massive movements while isolated movements result from stimulation of the posterior portion. At all points in the motor cortex stimuli above threshold evoked responses of the entire limb and often of the other ipsilateral limb as well, frequently with after-discharge as shown by clonic activity. Usually there was only a slight margin, often of no more than 0·4 volt, between a localized response and a generalized convulsion with all the features of a "cortical march" (*see also* Payne, Clark, Ward and Cowden, 1943).

An analysis of the responses elicited from various portions of the extremities (proximal or distal) yielded some rather surprising results in that in these experiments the distal portion of the extremity (forearm, wrist, hand and fingers; ankle, foot and toes) was involved in movements elicited by stimuli of threshold value or just above, less often than the more proximal parts of the extremities. Stimulation of 77 electrodes yielded clean-cut motor responses involving the head or extremities. Of these 21 gave head turning and in some instances movement of the proximal part of the extremities also occurred (but these are not counted below in movements of the extremities). 28 electrodes gave repeated specific responses in the proximal segments of the extremities while 18 gave similar responses in the distal segments. In ten instances stimulation of the electrodes with these weak stimuli gave a mixed response in both the proximal and distal segments of the extremities. In the unanæsthetized animal it would appear that there is not the predominance of representation in the cortex of the distal portion of extremities that was found with threshold stimuli in animals under anæsthesia by Boynton and Hines (1933). The situation appears to be different with further cephalization in the human subject (*see* Homunculus, p. 45; Penfield and Erickson, 1941).

The actual thresholds varied from 0·4 to almost 7 volts and appeared to bear little relation to the particular area stimulated. The total area over which electrodes were placed (fig. 2) was roughly divided into four portions: postcentral, central, anterior central and eye-fields. The postcentral group lay caudal to the central fissure, the central group comprised most of the "Betz" cell region, the anterior group, according to the map of McCulloch (1944) corresponded to area 6 but possibly included the strip area as well, while the eye-fields groups was the most anterior. The mean values of the thresholds obtained were 1·62 volts for the eye-fields, 1·61 volts for the anterior central group, 1·11 for the central group and 1·78 volts for the postcentral group. These averages are based on a total of 107 determinations, yet none of the differences between them is statistically significant. Since there were a few extremely high values (four in the postcentral group, three in the anterior group and two in the eyefields) which

would serve to increase inordinately the standard errors of these means, and, since these high values could easily have resulted from a localized hæmorrhage, &c., the means and "t" values (Snedecor, 1946) were recalculated after omitting the high values. The averages thus obtained are: eye-fields, 1·35 volts: anterior central, 1·08 volts; central, 1·11 volts, and postcentral, 1·29 volts. The difference between the means of the eye-fields and the anterior group after this omission just exceeds the value necessary for the 5 per cent level of confidence and none of the other differences is significant. The lowest threshold (0·4 volt) was found in the post-central region and the second lowest (0·5 volt) occurred in both the anterior central region and in the eye-fields.

(2) *Cortically induced sensation.*—While the usual response obtained was motion, responses were elicited from a few points which appeared to be due to sensory stimulation. The following protocol (Monkey 25, Electrode 8 post central)[1] is typical, except that in some other instances the monkey not only held up the hand and looked at it, but also rubbed it with the other hand.

Time	*Voltage*	*Duration*	*Response*
11:23	1	4	The monkey became violently active, turning and twisting his head and waving his left forearm. He continued to look at his hand for 20 seconds after the stimulus was discontinued
11:24	0·6	4	The monkey became restless at the end of the stimulus
11:25	0·8	4	With the stimulus the monkey began to wave his left forearm about violently, attempting to look at it over the neck board, and after the stimulus was over he stuck his hand up over the neck board and looked at it carefully for 15 or 20 seconds

Such responses, which are hard to interpret as other than sensory in character, with one exception (electrode lay on the anterior lip of the central fissure) were obtained from a rather small portion of the post-central region. The individual points are marked by solid triangles in figs. 1 and 2. Intermingled with or adjacent to these points there were motor or relaxation points and we can offer no explanation for this

[1]The protocols are usually copies of the transcribed records. In a few cases the wording was altered for the sake of greater clarity. The animal was seated, at the time of stimulation, in the usual examining chair with a rather large neck board. This made it impossible for the animal to touch the electrodes or their soldered connexions. There was some flexion at knee, hip, elbow and wrist. Where alterations were made in this initial position, the change is indicated by an italicized statement at the beginning of the response column. After the number of the electrode there is given the general location of the electrode as postcentral, central, eye-fields, or anterior central (anterior portion of motor cortex).

admixture. The violence of the response and the rubbing of the hand seem to bear a close resemblance to the original report of Bartholow (1874) but not to the usual description of stimulation of sensory points in the unanæsthetized human (Penfield and Boldrey, 1937). On the other hand, the restlessness with 0·6 volt may be an indication that our measures were crude and that a conscious patient would have reported sensation at this or even lower strength of stimulating current.

(3) *Comparison of the effects of strong and weak stimuli.*—The response to threshold stimulation was frequently not apparent in the complex response to stronger stimuli which elicited movement in several joints. Thus, in the following experiment, a simple fanning of the toes was seen with low voltage, but only violent flexion of the entire leg with higher strengths of current (Monkey 22, Electrode 4 postcentral).

Time	*Voltage*	*Duration*	*Response*
12:27	1	4	Questionable movement of the toes of the left foot
12:28	1·4	2	The left hind-leg was jerked violently into a flexed position in a tetanic manner. No clonus afterward
12:29	1	4	Simple fanning of the toes with the onset of the stimulus
12:30	1·1	4	Fanning of the toes throughout the stimulus

Fanning of the toes followed weak stimulation, but when the strength of the stimulus was increased, a different pattern of movement appeared. No fanning of the toes was seen, but there was a violent flexion of knee and hip. The difference in the responses was so marked as to make it improbable that this was a failure of observation and certainly fanning of the toes is not incompatible with flexion at knee and hip. One is forced to conclude that the response to 1·4 volt had a different pattern from that elicited by 1.0 volt.

In short, *strong stimuli may produce a different pattern of response in which the movement elicited by weak stimuli does not occur or is completely masked by other stronger movements.*

(4) *Cortically induced relaxation.*—A decrease in tone resulting from weak stimulation of the motor cortex of the cat was reported by Tower (1936). Stronger stimulation of the same points produced motion. A similar phenomenon was noted in the unanæsthetized monkey by Tower and Ward (1938). In the present series, we have seen four examples. They are indicated by the solid squares in the drawings of the various brains (fig. 1, Monkeys 21, 24, 25, and 29) and in the composite (fig. 2). In testing for alteration in tone one of us alternately extended and flexed the monkey's arms or legs while the other operated the stimulator. The

observer reported the onset and cessation of any change in tone without knowing when stimulation occurred. It will be seen in the first example (Monkey 29, Electrode 12 postcentral) that relaxation did not occur when the monkey's arms were well extended but was present in the left (contralateral to point stimulated) when stimulation occurred with the arms partially flexed. It was noted also that after a decrease in voltage from 1·8 to 1·6 no loss in tone was detected and that an increase from 1·8 to 2·0 produced a movement. In other words, the threshold effect was relaxation confined to the contralateral arm (and then only when the arm was partially flexed) while a stronger current led to an active motion.

Time	*Voltage*	*Duration*	*Response*
10:32	1	4	No observable effect
10:33	1·5	4	Questionable head movements
10:34	2·5	4	With onset of the stimulus the left arm was dropped and began a clonus which lasted, chiefly in the wrist, for about 6 seconds
10:35	2	4	With the onset of the stimulus the arm was retracted and partially extended but did not move a great deal, a clonus developed which lasted about 3 seconds after the stimulus
10:37	2	4	The arm was abducted slightly, apparently with a slight undulating clonus lasting a couple of seconds afterwards
10:39	1·6	4	No change of tone detected
10:40	1·8	4	With a 3 second latency there was evidence of reduction in tone in the left arm, none in the right
10:41	1·8	4	No evidence in change in tone with the extremities fairly well extended while being tested
10:42	1·8	4	At 2 seconds there was a relaxing of tension in the left arm to both flexion and extension
10:43	1·8	4	With the arms in an extended position, no change in tonus was detected

Reference to fig. 1 will show that this electrode was placed slightly caudad to the central fissure at about the junction of its upper and middle thirds, and in fig. 2 it will be seen that this one alone of several such placements produced relaxation.

The second example (Monkey 21, Electrode 3 anterior) differs markedly from the foregoing. Relaxation of some parts occurred simultaneously with motion elsewhere and neither response survived a slight (0·2) decrease in voltage. The presence of relaxation in both upper extremities at first and

the replacement of relaxation by movement in the contralateral arm seem as difficult to explain as is the greater ease with which relaxation appeared to be fatigued.

Time	*Voltage*	*Duration*	*Response*
10:31	1	4	As stimulus went on there was head turning to the ipsilateral side
10:33	1	4	There was head turning again to the ipsilateral side and closure of the contralateral (right) eyelid. There was a possible decrease in tone in both arms
10:34	0·8	4	No effect seen or felt
10:35	1	4	With the stimulus the head turned to the left again, both eyes closed and the tension both on extension and flexion in both upper extremities was reduced
10:37	1	4	There appeared to be very faint relaxation bilaterally in the upper extremities as the head turned around to the left
10:38	1	4	Possible slight relaxation of arm. The head turned to the left again
10:46	1	5	Good relaxation in the ipsilateral arm, some movement in the contralateral fingers and head turning to the ipsilateral side
10:48	1	4	The head turned to the ipsilateral side and there was movement in the right fingers of the hand with some tensing. Some clonus developed in the arm and was later seen in the neck
10:51	1	4	Again the head turned to the left and there was dorsiflexion of the right wrist. No difference in tone could be detected
10:53	0·9	4	The head turned to the ipsilateral side, nothing was seen or felt with respect to movement or reduction in tone in either extremity
11:00	1	4	With the stimulus the head turned to the ipsilateral side, there was tensing in the contralateral forearm and definite relaxation during the stimulus in the ipsilateral forearm
11:08	1	4	*Monkey on its back.* There was immediate relaxation in the left arm and movement in the right arm with head turning to the left

The electrode in this experiment was placed just caudad to the sulcus arcuatus and slightly ventral to the point where a continuation of the sulcus rectus intersects the arcuatus (*see* figs. 1 and 2).

Discussion.

Several points may be emphasized in the results obtained from these experiments. Firstly, relaxation is obtained from weak stimulation of the cortex of unanæsthetized monkeys of the kind reported in the cat by Tower (1936) and in the anæsthetized monkey and cat by Bosma and Gellhorn (1946, 1947*a*, 1947*b*). It is not to be confused with suppression, but rather, as stressed by Bosma and Gellhorn, it may be simply one of the phases of the response to cortical stimulation just as are reciprocal innervation and co-innervation. The relaxation observed as described in the two protocols in our experiments may be the same phenomenon, though the second case is not typical (as originally described by Tower, 1936) and its explanation is not clear. It is difficult to account for the change from the bilateral relaxation first noted to the ipsilateral relaxation with contralateral motion. A tegmental response (Hinsey, Ranson and Dixon, 1930) rather than a tonic neck response (Magnus and De Kleijn, 1912) might be the basis of this reaction. The shifting neural balance similar to that described by Graham Brown (1911) but applied to the negative side (relaxation or inhibition) of the picture rather than the positive (after-discharge) may be of importance especially since the animal was unanæsthetized. It is also difficult to account for the infrequency of relaxation with weak stimuli despite frequent placements of electrodes in almost identical positions (*see* fig. 2). One difficulty is the estimation of reduced tone in an animal that is not always quiet. Myographic recordings might give more consistent results. Bosma and Gellhorn (1946, 1947*a*, 1947*b*) show excellent figures of electromyographic records of this same phenomenon in the anæsthetized animal. They, too, found relaxation with weak stimuli and movement with stronger ones. It is regrettable that they did not compare their findings with those of Tower (1936) who reported eliciting relaxation from rather specific cortical areas.

It may be significant that we have not thus far seen evidence of "suppression" in the unanæsthetized animal like that described by Dusser de Barenne and McCulloch (1939) or A. A. Ward, Jr. (1948) (from area 24 in the monkey). It is further true that no report of suppression on cortical stimulation in the unanæsthetized human is to be found in the literature (Garol and Bucy in 1944 report suppression from area 4 in a patient during light ether anæsthesia). In the unanæsthetized cat, however, a stimulus (60 cycle sine wave current lasting 4 seconds) directed into the white matter of the gyrus cingulus brought the animal to a halt for the duration of the stimulus with one foreleg poised off the table. No sinking of the animal was noted which could have been interpreted as a melting of tone in the extremities or body musculature (J. W. Ward, un-

published observation). Whatever is the explanation for these differences, these various observations certainly raise the question as to the relationship of these phenomena to one another and to their significance in the total functional organization of the nervous system.

In experiments conducted under general anæsthesia, the threshold of the Betz-cell area is reported to be significantly lower than that of other fields (Walshe, 1942: Fulton, 1946, and others). With the unanæsthetized animal, as shown above, there appears no such difference in excitability of the different regions tested. The thresholds of the most excitable points in all areas were equally low and the difference between the mean values not statistically reliable. However, after omission of certain high values, the mean value for the eye-fields became reliably higher (to the 5 per cent level of confidence) than that of our anterior central group (largely what has been termed area 6) but *not* significantly higher than that of the central (area 4) or the postcentral group. These results are in accord with those reported by Penfield and Boldrey (1937) who found no difference in threshold between areas 4 and 6, and an excitability of the postcentral region even greater than that of the precentral in conscious human patients. These observations suggest that the anæsthesia usually employed during cortical stimulation experiments alters the threshold of cortical regions specifically, an observation reported for the barbiturates by Bucy (1942) based on clinical observations in athetosis in man.

The purely motor responses observed in these experiments on unanæsthetized animals with threshold stimuli ranged from isolated movements of the hip or shoulder to flexion of single joints in the fingers. We saw no predominant frequency or ease of elicitation of movements that occurred either in proximal or distal portion of the extremity. This situation appears to be at variance with the observations of Boynton and Hines (1933) but in agreement with the findings on the cat, rabbit and monkey of Murphy and Gellhorn (1945). The basis for this difference is not clear.

Finally, a word should be said about the stability of response elicited from points in the cortex in these experiments. As has been reported previously in acute and chronic experiments (Ward and Clark, 1935; Clark and Ward, 1937, and Ward, 1938) when sufficient time is allowed between stimuli and the electrodes are fixed on the cortex so that they cannot move from stimulus to stimulus, a cortical point yields a constant response with stimuli of constant values. These observations are valid in the unanæsthetized monkey, according to our results, so long as the same initial position of the limb is maintained with each stimulus. These findings are in keeping with those observed in the unanæsthetized cat (Ward, 1938).

SUMMARY.

(1). On the basis of the threshold of the responses elicited from the cortical areas 4 and 6 it is not possible to distinguish them apart in the unanæsthetized monkey.

(2). The response to a strong stimulus of a cortical point may mask the details of a simpler response elicited by weaker stimuli.

(3). Relaxation with stimuli under threshold values for motor responses from the cortex were obtained in the unanæsthetized monkey.

(4). No predominance was found in the frequency of responses to cortical stimulation of proximal and distal segments of the extremities of the unanæsthetized monkey.

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